of this document. Again, these factors are irrefutably implicated in MSD development and recovery, as factors that modify the body's response to external risk factors and its ability to recover from insult. But their presence in the equation of etiology does not remove the primary necessity to identify and control external, workplace-based risk factors.

Reparative Capacity of Individuals. The physiological effects of the risk factors and modifiers presented in Section D are themselves modified by the worker's individual capacity to accept and repair the damage caused. This capacity may be likened to the ability of the body to process a chemical exposure. Depending on the body's defenses, a given atmospheric concentration of toxin will result in cells and tissues receiving a particular dose of the toxin. Over time, this dose, modified by the body's capacity to detoxify and/or clear the substance and its metabolites, will result in a measurable body burden.

Although the analogy is simplistic, and other disease mechanisms are probable, it is possible to visualize certain effects of biomechanical risk factors through this model. An exposure to a biomechanical risk factor of given intensity, duration, and temporal profile can result in an internal ''dose'' that makes demands on the body's reparative capacity for ''detoxification'' of the dose. The cumulative trauma model suggests that the resultant ''body burden'' may be seen as partly the result of exposure and repair capacity. Armstrong *et al.* (1993) proposed a model (called a ''cascade'' model) of this process that also incorporates a staged series of challenges to the body. The body's response to a particular biomechanical ''dose'' can itself generate new physiological or anatomical stressors; the effectiveness of the body's response to these new stressors also depends partly on individual capacity. Likewise, pre-existing or underlying disease can also compromise reparative capacity as well as predisposing tissues to further injury.

The components of individual reparative capacity include:

- Genetic factors. These include basic inherited characteristics of the individual, such as body dimensions (anthropometry), physiological variables, and gender. Genetically based personal differences include variation in bone length and tendon attachment points (which affect the mechanical advantage of a muscle in a given posture), muscle mass and distribution of fiber types, laxity of ligaments, intervertebral disk cross-sectional area and nucleus fluidity, tendon size, and carpal tunnel size (Radwin and Lavender, NRC 1998, Ex. 26–37).

Gender may be seen partly as representing anatomical and physiological differences among workers (see summary in Faucett and Werner, 1998, Ex. 26–425). Women's anthropometry may not fit many jobs designed originally for the average male. It is important to understand, however, that gender is also a surrogate for a large complex of social and economic differences among workers, as well a differences in exposure between males and females. Many of these differences influence patterns of disease and recovery (Messing, Chatigny, and Courville, 1998a, Ex. 26–566; Messing *et al.*, 1998b, Ex. 26–300).

- Acquired characteristics. Acquired characteristics include physical conditioning, previous or concurrent disease status, and the effects of aging. The aging process is strongly influenced by both genetic and acquired characteristics. In any case, OSHA's mandate to assure a safe and healthy workplace is not limited to workers below an arbitrary age threshold but encompasses workers of all ages. Acquired characteristics can modify some genetically based characteristics. For example, type and intensity of exercise can alter muscle mass and fiber type distribution. Likewise, a worker's level of skill and work habits can substantially affect the impact of biomechanical stressors on body tissues.

It is important to recognize that the effects of risk factors and modifiers found in the work environment are modified at the individual level by these personal factors. However, the primary purpose of job analysis and workplace interventions is to make work safe for as many workers as possible. Hence, this document considers the measurement, characterization, and reduction of work environment risks and modifiers to be the most important objective of the ergonomics program rule.

Work Techniques and Skill Level. Personal factors also include work technique and skill level. In some situations, the predominant factors influencing MSDs are individual anatomy, work style, posture, and technique. For example, the well-recognized upper extremity disorders of sign language interpreters (Feuerstein and Fitzgerald, 1992, Ex. 26–1284), or the hand problems of musicians (Amadio and Russotti, 1990, Ex. 26–925; Fry, 1986, Ex. 26–850), are usually addressed on an individual (intrinsic) basis, because either no tool is involved, or the potential for tool modification is limited.

Other situations clearly preclude addressing problems on an individual basis. For example, the vascular and neurologic problems produced by hand-arm vibration occur with such high attack rates and predictability that an effective control strategy necessarily addresses the tool and extrinsic exposure rather than individual susceptibility (Pyykko 1986, Ex. 26–662). In some industries, such as meatpacking, hand and wrist problems have been so prevalent and associated so strongly with particular tasks that identifying cause in a work process is unambiguous (Schottland *et al.*, 1991, Ex. 26–1001; Masear, Hayes, and Hyde, 1986, Ex. 26–983).

In still other settings, the multi-dimensional pattern of personalized risk factors, non-work risk factors, and external, work-related risk factors complicates etiology identification. As with other chronic and sub-chronic diseases, it may be difficult, and sometimes impossible, to differentiate between underlying morbidity and causative, exacerbating, or even disabling features (stressors) in the external environment.

3. Medical and Diagnostic Issues

The development of an ergonomics standard for U.S. workplaces poses specific challenges for disease identification. The relationship between MSDs and exposure to even well-recognized risk factors, such as heavy repetitive lifting and hand-arm vibration, poses different sets of challenges for the recognition of exposures and their control than has been the case for many more traditional workplace exposures and disorders. The inhalation of asbestos fibers, for example, has well-defined and accepted endpoints, such as lung cancer and mesothelioma, and intermediate health effects at the tissue or cellular level are less important objects of dust control. Formaldehyde and other irritants have immediate and recognizable effects on mucosa, so that overexposure is often obvious, and the parameters of acute effects and detection thresholds all fall within a limited range of measurements. Physical hazards such as noise and radiation are highly organ-specific or have universally accepted risk profiles. For such hazards, exposure assessment does not require significant attention to individual work factors or personal factors, or there may be a consensus test for disease (as for noise).

For MSDs, on the other hand, microanatomic injury and repair is often sub-clinical and generally invisible to clinical testing or surveillance measures. Although, the object of much active research, the relationship between sub-threshold injury and the onset of recognized clinical disorders is imprecisely understood. Because of regional and individual differences in diagnosis and treatment, disease recognition depends on professional practice, diagnosis, and treatment patterns.

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B. Biomechanical Risk Factors and Modifiers

1. Overview

This section has two purposes:

- To present a framework for and classification of major observable and quantifiable workplace risk factors for neuromuscular and musculoskeletal disorders (MSDs).
- To define and explain these risk factors and to briefly explore possible mechanisms by which exposure to these stressors could cause MSDs.

The section begins with a summary exploration of the issues involved in establishing a causal relationship between aspects of the work environment/process and musculoskeletal disorders (Section B.1). It then presents the classification scheme used in the section, with brief reference to possible mechanisms of effect. Sections B.2 and B.3 present current knowledge of the basic physical risk factors and modifying factors identified by epidemiological and laboratory research.

a. Epidemiological Criteria for Establishing Causation. Good epidemiology requires accurate and consistent identification and quantification of both exposure and outcome. In the rapidly evolving fields of research relevant to MSD etiology, there are still problems with measurement, quantification, and even recognition of particular risks and disease outcomes. However, the research referenced in this document demonstrates substantial agreement over a wide range of research methodologies concerning the causal association between a set of commonly recognized stressors and MSD outcomes.

The risk factors discussed in this section have been shown to cause or contribute to MSDs, in accordance with generally accepted criteria for assessing a cause-effect relationship.

The following list of such criteria (based on Hill, 1965, Ex. 26–376; Hennekens, Buring, and Mayrent, 1987, Ex. 26–428; Bernard and Fine, 1997, Ex. 26–1; Rothman and Greenland, 1998, Ex. 26–870) is not exhaustive but represents consensus in the field of epidemiology. Note that, with the exception of temporality, none of these criteria is a necessary or sufficient basis for determining causality: the absence of any criterion other than temporality in a study does not necessarily invalidate a causal hypothesis. But the presence of each factor, while not proving causality, does strengthen that hypothesis. Any given study may not satisfy each criterion, but the cumulative burden of evidence, from the many studies cited in this document, strongly argues for a causal relationship between the risk factors presented in this section and MSDs. These criteria are:

- The strength of the association. The larger the association, the less likely is an interpretation invoking undetected bias or unmeasured confounders. If bias or confounding are operative, they would have to be of a larger magnitude to explain the size of the association, making it less likely that the study would have overlooked them.
- Biological plausibility. Knowledge of a known or understandable proposed mechanism aids determination of causality.
- Consistency with other research. Similar results from independent studies, especially with different measurement techniques, strengthen a causality hypothesis.
- Temporality or appropriate time sequence. The proposed exposure (the risk factor) should be present prior to the proposed effect or outcome (here, indicators of MSDs).
- Dose-response relationship (biologic gradient). If higher levels of exposure are associated with higher levels of outcome, this can indicate causality. However, a causal relationship may exist but be hidden by a non-linear dose-response relationship. The presence of a dose-response relationship can also indicate a confounder with its own biologic gradient.

A sixth criterion, specificity of association, is often added to this list. This term refers to the degree to which a particular outcome is always associated with a particular risk factor. Because of the overwhelming evidence for multifactoral causation of MSDs, the specificity of association is low for most risk factors and musculoskeletal outcomes (Kourinka and Forcier, 1995, Ex. 26–432). Thus, this criterion is generally not useful in assessing causality in MSD etiology (with the possible exception of the specific association of vibration exposure with neurovascular disorders in the hands). In general, a specific risk factor can be associated with a number of different outcomes.

b. Classification of Risk Factors and Modifiers. As much as possible, the risk factor classification employed in this document uses the definitions and concepts defined by NIOSH in the publication, ''Musculoskeletal Disorders and Workplace Factors'' (Bernard and Fine, 1997, Ex. 26–1), combined with definitions and concepts developed in the draft ANSI ergonomics standard, Z–365 (1998, Ex. 26–1264). This discussion separates the risk factors into two basic families of concepts: basic risk factors and modifiers. The basic risk factors presented here are the aspects of work that most researchers agree cause or exacerbate MSDs. The modifiers are characteristics of a specific exposure to a risk factor that may affect the level or type of strain produced within tissues. Although there is a growing body of evidence linking psychosocial and work organization factors with the development of MSDs, those factors are not addressed here (other than the obvious impact of work organization on work pace). The following sections focus on the biomechanical or physical risk factors:

- Basic Biomechanical Risk Factors (Section B.2):

—Force
—Awkward Postures
—Static Postures
—Repetition
—Dynamic Factors
—Compression
—Vibration

- Modifying Factors (Section B.3):

—Intensity
—Duration
—Temporal Profile
—Cold Temperatures

Other classification systems are possible and valid. For instance, Kourinka and Forcier (1995, Ex. 26–432) present a broader system that links force, repetition, and duration as components of ''musculoskeletal load.'' Radwin and Lavender (in NAS, 1998, Ex. 26–37) and the ANSI draft standard Z–365 (1998, Ex. 26–1264) prefer to list repetition as a modifier or ''characteristic property'' rather than as a basic risk factor. The system used here represents one useful classification scheme; the component terms maintain essentially the same definition in any of the frameworks currently in use. Most importantly, these differences in classification are relatively trivial and do not affect the evidence showing that all of these factors are implicated in the etiology of work-related MSDs.

2. Basic Risk Factors

This section details the definitions, measurement issues, and some of the proposed effect mechanisms associated with basic biomechanical risk factors. No attempt is made to prioritize risk factors by importance, because the relative contribution of each stressor to MSDs depends on the particulars of the work environment and task structure, including the presence or absence of other risk factors. For instance, Radwin and Lavender (in NAS, 1998, Ex. 26–37) note that for a primarily static task, postural risks merit the closest attention in job analysis, while a dynamic manual material handling job requires more attention to dynamic stressors, such as range of motion, velocity, and acceleration of movement. Evidence for the relationship between these risk factors and MSDs is presented in detail in Section V.C of this preamble and the Appendices (Ex. 27–1). This section provides only cursory treatment of the mechanism of tissue injury attributable to these risk factors; Section V–C presents this aspect of MSD etiology in detail.

a. Force. Force is the mechanical effort required to carry out a movement or to prevent movement. Force may be exerted against a work piece or tool, or against gravity, to stabilize body segments. Force does not necessarily imply motion. The dynamic act of lifting a work piece and the static act of holding that work piece in position both require force, generated by muscles, transmitted through tendons, and exerted by body segments on the work piece. In determining the risk posed by force requirements of the task, it is useful to consider muscle force and output force of body segments separately.

Muscle Force. Muscle force is the actual mechanical effort exerted by the combined contraction of muscle fibers. The total force generated by any one muscle is a function of many factors, including the cross-sectional area of the muscle, the length of the muscle during contraction (*i.e.*, where the length range falls between full contraction and

full extension), and the degree of fatigue. Research generally characterizes muscle force by surrogate measures of muscle activity (*e.g.*, amplitude of electromyographic [EMG] signals, generally expressed as a percentage of the amplitude measured at maximum voluntary contraction [MVC]). Because of the electrical activity associated with muscle contraction, muscle force is the most easily measured aspect of tissue involvement. But full characterization of potential tissue damage requires attention to all links in the pathway through which muscle force is transmitted to output force (Section 2.a). Thus, force requirements affect tension on tendons (which transmit muscle force to bones), shear force, friction, and irritation induced by lateral forces on tendons and tendon sheaths (as they are pressed against surrounding anatomical structures) and the strain at the insertion of tendons on bones.

Estimating muscle force from external characteristics of the task can be complicated compared to measuring muscle activity (such as taking EMG measurements with deep wire electrodes implanted directly in the muscle fibers of interest). First, many external job characteristics can affect muscle force requirements, and some of these characteristics may not be recognized in a job analysis. For example, Kourinka and Forcier (1995, Ex. 26–432) note several factors that affect muscle force required for a grip: presence of other risk factors (such as awkward postures required by grip type and handle size), the coefficient of friction of the work piece surface, whether gloves are required, and individual variations in technique.

Second, the lever arm (the distance from point of force application to the fulcrum—the joint center) for most muscles is generally much smaller than that of the external load (Radwin and Lavender, in NAS, 1998, Ex. 26–37). This means that muscle forces are usually several times greater than the external load. Thus, accurate modeling requires precise estimation or modeling of actual lever arm lengths.

Third, fatigue affects muscle fiber recruitment patterns within a single muscle, as well as recruitment (substitution) patterns of alternative muscles (Parnianpour *et al.*, 1988, Ex. 26–1150). When secondary muscles are recruited to assist a fatigued primary muscle, the recruited secondary muscles may be more vulnerable to injury due to less-advantageous lever arm length, smaller size, or less-than-optimal fiber length in the work posture (see Section 2.b).

Despite these difficulties, modeling approaches can often predict internal force requirements accurately. For instance, Marras and Granata (1997a, Ex. 26–1380) showed that measured pressures in the L5S1 intervertebral disk generally match values predicted by modeling. (Internal disk pressure is a result of forces exerted on the disk by muscles and gravity.)

Output Force. The force exerted by body parts to move or hold the work piece (often against gravity) is obviously a function of muscle force. However, the relationship is strongly affected by other variables, the most important being posture. Deviations from a so-called ''neutral posture'' (see Section 2.b) can dramatically reduce the amount of muscle force translated into output force. The ''lost'' force is generally seen in inefficient coupling of the contractile proteins in muscle fibers or in force exerted by muscles and tendons against adjacent anatomical structures as the force transmission changes direction. In addition, most holding and moving tasks involve input from several muscles, often working in opposition. Skilled, small-motor activities involve co-contraction of antagonist muscles to generate precisely graded movements, joint stabilization, or holding forces. Thus, substantial muscle activity can be associated with very little net output force. In addition, these co-contractile forces act additively on the joint components (ligaments, cartilage, and bone). For the researcher, this has important implications. For example, measurements of the weight of a work piece or the finger forces necessary to move a computer mouse may substantially underestimate the potential damage to the muscles, tendons, joints and other soft tissues involved.

Guidelines for manual materials handling (*e.g.*, Snook and Ciriello, 1991, Ex. 26–1008; NIOSH, 1981, 1994, Exs. 26–393 and 26–572) clearly note that the weight of the load, in isolation, is not a sufficient measure of musculoskeletal stress.

b. Awkward Postures. This risk factor is generally conceptualized as postures deviated from a neutral position. In this document, ''posture'' means the angle between two adjacent body segments. A so-called ''neutral posture'' angle can be determined for each joint. This term seems to suggest the resting position of the joint, but it actually encompasses two biomechanical criteria necessary for optimal development of muscle force:

- The biomechanical relationship of the two body segments that presents the largest lever arm upon which the muscle force acts.
- The length of the muscle that allows it to develop the greatest force most rapidly. For most muscles, the physiological and physical relationships between the two contractile proteins, known as the length-tension and the length-velocity relationships, mean that maximum force and speed of contraction can be developed when the muscle is in a position between greatest extension and greatest contraction.

However, the term ''non-neutral posture'' should only be seen as a first approximation of a stressful, awkward posture, for several reasons. First, neutral posture is generally defined in terms of muscle length, although joint angles have implications for other tissues: what is optimal for one tissue may not be the optimal joint angle for another. For example, a roughly 90-degree elbow angle satisfies both criteria above for optimal biceps activity. But that posture may stretch the ulnar nerve against the elbow, suggesting that a more open elbow angle is necessary for optimal nerve function and safety.

Second, most body exertions involve more than one muscle, each of which may be in optimal biomechanical and length relationship at a different joint angle. Third, the body can adopt postures that are not necessarily the optimal biomechanical or length-tension relationships for muscles, but that result in the lowest sum of muscle activation to stabilize body parts against gravity.

Fourth, non-neutral postures are sometimes defined in relation to their association with tissue damage, not to a biomechanically sub-optimal joint angle. For example, a 90-degree abduction of the upper arm may put some shoulder muscles (*e.g.*, the deltoids) in a relatively ''neutral'' posture, but can expose the brachioplexus to compressive forces from other muscles and anatomical structures. This posture can also entrap the tendon of the supraspinatus muscle between the acromion and the head of the humerus (Hagberg, 1984, Ex. 26–1271). To fully characterize the degree to which a posture is ''awkward,'' it is necessary to take an integrated overview of the tissues involved, defining which muscles and other tissues are involved in the position and what the implications are for tissue damage.

With these concerns in mind, Kourinka and Forcier (1995, Ex. 26–432) separate the term ''awkward postures'' into

three concepts, which may characterize a particular posture in combination or alone:

- Extreme postures. This term is used in the NIOSH review of epidemiological evidence (Bernard and Fine, 1997, Ex. 26–1). Extreme postures are joint positions close to the ends of the range of motion. They require more support, either by passive tissues (*e.g.,* ligaments and passive elements of the muscles) or increased muscle force. These positions may also exert compressive forces on blood vessels and/or nerves. Note, however, that some joints, such as the knee, are designed to be used close to the range-of-motion extremes.

- Non-extreme postures that expose the joint to loading from gravitational forces, requiring increased forces from muscles and/or load on other tissues. For instance, holding the arm at 90 degrees to the body does not represent an extreme posture in terms of muscle length. But the position allows gravitational forces to exert a pull requiring roughly 10% of maximal strength from the associated muscles (Takala and Viikari-Juntura, 1991, Ex. 26–1014).

- Non-extreme postures that change musculoskeletal geometry, increasing loading on tissues or reducing the tolerance of these tissues. This third factor includes the reduction in available lever arm for muscles, described above. An example of increased loading is provided by experiments (Smith, Sonstegard, and Anderson, 1977, Ex. 26–1006) demonstrating that even non-extreme wrist flexion can press the finger flexor tendons against the median nerve. Experiments by Adams *et al.* (1980, Ex. 26–701) indicate that combined flexion and twisting or bending of the spine reduces tissue tolerance of the intervertebral disks, predisposing them to rupture.

c. Static Postures. Static postures—postures held over a period of time to resist the force of gravity or to stabilize a work piece—are particularly stressful to the musculoskeletal system. More precisely, static postures are usually defined as requiring isometric muscle force—exertion without accompanying movement. Even with some movement, if the joint does not return to a neutral position and continual muscle force is required, the effect can be the same as a non-moving posture. Since blood vessels generally pass through the muscles they supply, static contraction of the muscle can reduce blood flow by as much as 90%. The consequent reduction in oxygen and nutrient supply and waste product clearance results in more rapid onset of fatigue and may predispose muscles and other tissues to injury. The increased intramuscular pressure exerted on neural tissue may result in chronic decrement in nerve function. The viscoelastic ligament and tendon tissues can exhibit ''creep'' over time, possibly reaching failure thresholds beyond which they are unable to regain resting length.

d. Repetition. Appendix I lists repetition as a basic risk factor. This section follows that categorization. However, repetition can have characteristics of both a basic risk factor and a modifier (the ANSI draft standard, Z–365, 1998, Ex. 26–1264, gives repetition modifier or ''characteristic property'' status). High repetition may act as a modifying factor, exacerbating the basic risk factors of force and posture. But high repetition also may have its own tissue effects (combined with the dynamic factors described in Section 2.e). For example, increased friction-induced irritation of finger flexor and extensor tendons in their sheaths can result in tendinitis and lead to increased pressure in the carpal canal. A moderate level of repetition can be seen as protective, since it can increase muscle strength and flexibility (this is the concept behind exercise). It can also assist blood flow through muscles, thus relieving the stressful nature of static muscle contractions. Ideal work cycles keep overall repetition rates in a middle zone between the injurious extremes of static contraction and excessive repetition.

e. Dynamic Factors (Motion). Motion of body segments consists of both linear motion and rotational motion around a joint. Present research addresses the effects of kinematic measures of posture: both angular and linear velocity (speed of motion) and acceleration (rate at which velocity increases or decreases). It is possible that, to a degree, measured acceleration and velocity are surrogates for increased force and postural risk factors. For example, Marras and Granata (1995, 1997b Exs. 26–1383 and 26–169) find that increased velocity and acceleration in trunk lateral bending and twisting result in measurable increases in both compressive and shear forces experienced by the intervertebral disks. But dynamic factors themselves may result in increased tendon travel and irritation. Viscoelastic soft tissues, such as tendons, spinal discs, and ligaments, have a fixed, intrinsic capacity to regain resting dimensions after stretching. Brief movement cycles may involve peak accelerations that can exceed tissue elasticity limits during an otherwise moderate task. The biodynamic literature suggests that, even in tasks performed for a short time, the acceleration and velocity of movements may pose risks that would not be predicted by the muscle forces or joint angles alone.

f. Compression. Compression of tissues can result from exposure that is external or internal to the body. Depending on the tissue compressed, the effects are manifested in quite different ways (see Section V–D of this preamble).

External Compression. Moderately sharp edges, such as tool handles, workbench edges, machine corners, and even poorly designed seating, concentrate forces on a small area of the anatomy, resulting in high, localized pressure. This pressure can compress nerves, vessels, and other soft tissues, resulting in tissue-specific damage (*e.g.,* degraded nerve transmission, reduced blood flow, and mechanical damage to tendons and/or tendon sheaths). These changes may themselves result in disease or predispose other tissues to damage.

The most common sites for compression MSDs are in the hands and wrists. Since natural selection has resulted in well-developed, padded gripping areas on the hands (in particular, finger pads and the thenar and hypothenar pads on the palm), injury is most often seen outside these areas: the sides of the fingers, the palm, and the ventral side of the wrist. For instance, the prolonged use of scissors can cause nerve damage on the sides of the fingers. Compression MSDs have also been identified in the forearm, elbow, and shoulder.

Internal Compression. Nerves, vessels, and other soft tissues may be internally compressed under conditions of high-force exertions, awkward postures, static postures, and/or high velocity or acceleration of movement. For example, strong abduction or extension of the upper arm, as well as awkward postures of the neck, can compress parts of the brachioplexus under the scalene muscles and other anatomical structures. This compression can result in nerve and/or blood vessel damage or in eventual damage to the tissues served by these nerves and vessels.

There are other sources of internal compression, also the secondary result of exposure to other risk factors noted in this document. Examples include:

- Intramuscular pressure developed during forceful contraction. (This is the main mechanism resulting in

compression of blood vessels internal to the muscles during static contraction).

- Pressure due to reparative swelling of tissues injured in work processes. (For example, the inflammatory swelling of flexor tendon synovial sheaths, in response to friction and irritation, can increase pressure in the carpal tunnel and compress the median nerve.)

g. Vibration. Vibration is normally divided into two categories:

- Segmental vibration or vibration transmitted through the hands. Segmental vibration appears to damage both the small, unmyelinated nerve fibers and the small blood vessels in the fingers, resulting in two specific diseases: vibration-induced white finger (VWF) and vibratory neuropathy. Together, these are called the hand-arm vibration syndrome (see below). Segmental vibration has also been implicated in carpal tunnel syndrome.

- Whole-body vibration, or vibration transmitted through the lower extremities and/or the back. Whole-body vibration is implicated in low back disorders and a host of less well-understood symptoms.

Recent research suggests that vibration should be further subdivided into two types:

- Harmonic or oscillatory vibration (due to a constant driving source, such as a grinding wheel or holding a powered tool such as an electric drill)

- Impact vibration (due to single impact, such as hammering a nail)

- Percussive vibration (bursts of separable impacts, such as those produced by a pneumatic riveting tool or a jackhammer)

It is possible that the thresholds for effects of these three types of vibration are quite different, with impact and percussive vibration having physiological effects at much lower measured exposure times.

Three classes of effect due to vibration are discussed in Section V–D and the Appendices (Ex. 27–1):

- Vascular damage, leading to premature vasoconstriction and insufficient circulation in the fingers. These effects give rise to the original name for occupationally induced Raynaud's syndrome: vibration-induced white finger (VWF). In 1987, a consensus panel, meeting in Stockholm, coined the term hand-arm vibration syndrome (HAVS) to give equal weighting to neurological symptoms (Gemne *et al.,* 1987, Ex. 26–624).

- Neurological effects. These effects involve damage to both the median nerve and to the small, unmyelinated nerve fibers in the fingers.

- Musculoskeletal effects. Kourinka and Forcier (1995, Ex. 26–432) list a number of possible effects in this category, including impaired muscle strength and osteoarthrosis of some upper extremity joints.

Finally, some research suggests that vibration received aurally (*i.e.*, noise) can, itself, result in increased static muscle loading (Kjellberg, Sköldström, and Tesaiz, 1991, Ex. 26–432).

3. Modifying Factors

This section elaborates on the definitions and measurement issues associated with the classification of modifying factors presented in Section B.1. Evidence for the relationship between these modifying factors and MSDs is presented in Section C. The following measures are not risk factors in themselves; rather, they modify the effects of the basic risk factors. To fully characterize exposure, investigators measure both the basic risk factors and the relevant modifiers.

a. Intensity or Magnitude. Intensity or magnitude is a measure of the strength of each risk factor: how much force, how deviated the posture, how great the velocity or acceleration of motion, how much pressure due to compression, how great the acceleration level of vibration, etc.

b. Duration. Duration is the measure of how long the risk factor was experienced. This is a task-specific measure and is generally combined with a comprehensive, job-specific characterization of the temporal profile of the exposure (Section 3.c). Frequency and duration are related, *i.e.*, the more frequently a task is performed, the greater the duration of exposure.

c. Temporal Profile (Recovery Time and Pattern of Exposure). The combined effects of the basic risk factors, modified by intensity and duration, tax the recovery and repair capacities of the body. Recovery capacity is strongly related to the time available for tissue repair. Thus, accurate exposure assessment takes into account the way that risk factors vary over time. Excessive metabolic load and inadequate rest schedules deprive the body of recovery time to accomplish repair on strained tissues. The pattern of exposure can be as important as total magnitude or cumulative exposure. For instance, a cumulative exposure duration of 4 hours, spread over two 8-hour work days, can be associated with substantially different health effects than a single, one-time exposure of 4 hours. Kourinka and Forcier (1995, Ex. 26–432) note that assessment of temporal profile would include:

- Task variation over a given time period (hour, day, week)

- Characteristics of the duty cycle: the proportion of the task in which stressors are high, compared to when they are low

- Schedule of micropauses (of a few seconds) every few minutes

- Distribution of formal rest breaks

- Shift and overtime schedules

d. Cold Temperatures. Cold is a well-established exacerbating factor in the development of vibration-related disease. In addition to aggravating pre-existing disease and injury, cold environments compromise muscle efficiency. Cold-related injuries to the hands result in several vascular and neurological disorders. Perhaps the most common effect of cold is its ability to reduce cutaneous sensory sensitivity and thus compromise manual dexterity. Workers with cold-desensitized fingers may grasp loads with more force than necessary, due to reduced sensory feedback, thus exposing muscles, soft tissues, and joints to increased tensile and compressive forces.

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C. Evidence for the Role of Basic Risk Factors and Modifying Factors in the Etiology of MSDs

This section summarizes the extensive body of evidence for the involvement of workplace stressors in musculoskeletal disorder (MSD) causation. For each of the basic risk factors and modifying factors described in Section V–B, this section presents highlights from the relevant epidemiological, laboratory, and psychophysical studies, as well as a summary of the evidence. Section V–D and the Appendices (Ex. 27–1) explore this body of evidence in much greater detail.

1. Quality of the Evidence

The evidence from epidemiologic, laboratory, and psychophysical studies in the Health Effects Section supports a causal relationship between workplace stressors and MSD outcomes. The proposed mechanisms of effect, detailed in Section V–D of the preamble and in the Appendices (Ex. 27–1), support the biological plausibility of the link between stressors and disease—one of the five criteria useful in establishing causality (see Section B.1.a). These criteria require attention to population studies relating exposure and effect (epidemiology), to physiological measurements that show a plausible mechanism for disease causation or exacerbation (laboratory studies), and to subjective perceptions of fatigue and pain (psychophysical studies).

The epidemiological studies in this field have been criticized because they tend to feature cross-sectional research design and rely on worker self-reports. These studies may have an increased risk of common-instrument bias (if based on self-report) and present obstacles to determining causality, due to their inability to establish temporality. The NIOSH review discussed below (Bernard and Fine, 1997, Ex. 26–1) selected the studies with the best design and further weighted these studies' contributions to the review's conclusions by methodological quality. Still, some investigators feel that NIOSH was not exclusive enough in its selection of acceptable studies. (Note that although Gerr [1998, Ex. 26–426] makes this criticism in the NAS symposium [1998, Ex. 26–37], he also states that he doubts whether the exclusions he suggests would make a substantial difference in the overall conclusions NIOSH reaches about work-relatedness.) NIOSH notes that ''The document represents a first step in assessing work-relatedness of MSDs.''

It is useful, however, to look more deeply at the criticisms of self-reported studies. Punnett (1998, Ex. 26–442) reviews the wide variety of studies that demonstrate the validity of self-report measures. These studies further suggest that common-instrument bias (the notion that a worker's perception of high exposure might lead him/her to report higher symptom status, or vice versa) may pose less of a problem than critics suppose. Punnett notes that a number of well-designed keyboard studies found differences between self-reported and observed keying times, but these differences were non-differential between cases and controls. Symptom status, in other words, did not bias overall reporting of exposure one way or the other. The NIOSH summary of epidemiological evidence for low-back MSDs (Bernard and Fine, 1997, Ex. 26–1) does not support the assumption that self-reported bias inflates associations. Of the 13 studies (out of 18 reviewed) with a positive relationship between work-related lifting and forceful movements, those relying on objective measures of exposure showed higher odds ratios (ORs) (2.2–11) than those relying on subjective measures (1.2–5.2).

Likewise, looking at objectively measured as opposed to self-reported MSD outcomes, self-reported symptoms do correlate with objectively measured disease. Bernard *et al.* (1993, Ex. 26–439), for example, found that when compared to non-cases for increased median nerve latency, subjects defined as CTS cases on the basis of self-reported symptoms showed an OR of 42.5 (with a wide 95% CI: 1.61–1122, due to small sample size).

Although other types of bias are difficult to detect in cross-sectional studies, when they occur they are likely to underestimate rather than overestimate the relationship between exposure to stressors and disease. For instance, the ''healthy worker'' bias, the preferential departure of symptomatic workers from high-exposure jobs, artificially lowers the disease prevalence in these jobs, reducing the calculated association of stressor exposure to MSD in analysis. The clear association noted by the NAS report (1998, Ex. 26–37) between MSDs and jobs with high physical load is thus derived despite the effect-reducing influence of the ''healthy worker'' bias. This example also demonstrates that a researcher can make plausible hypotheses about the direction of effect in some cross-sectional studies. It is highly unlikely that workers experiencing MSD symptoms would preferentially transfer into jobs with higher physical exposure (which would artificially elevate epidemiological estimates of effect). It has, in fact, been shown that symptomatic workers do tend to leave jobs that have high levels of MSD risk (Punnett, 1998, Ex. 26–442). Silverstein *et al.* (1988, Ex. 26–1004), in a follow-up study at one of the plants examined in their earlier studies, found that those subjects in the high-force/high-repetition exposure category who were symptomatic in the original study were no longer in that exposure category at the time of follow-up.

This section does not evaluate the growing body of intervention research relating reduction in the number and severity of MSDs to intentional reductions in exposures. However, the recent NIOSH study of MSDs and workplace factors (Bernard and Fine, 1997, Ex. 26–1) includes studies that demonstrate a reduction in disease as a result of interventions that reduce exposures. Goldenhar (1994, Ex. 26–126) and Smith, Karsh, and Moro (1998, Ex. 26–445) carried out reviews of the intervention literature. While noting the potential value of intervention research, both reviews note substantial deficits in research sample size and study design. Despite these drawbacks, Smith, Karsh, and Moro find evidence for the injury-reduction potential of redesigned hand tools, weight-handling devices (*e.g.*, hoists, articulated arms), and other work station alterations, as well as exercise and training. The General Accounting Office study (1997, Ex. 26–5) of ergonomic program effectiveness (focusing on five case studies) found that successful programs were based on a core set of elements: management commitment and employee involvement, identification of problem jobs, development of solutions, training and education, and medical management. Programs based on these elements showed reductions in injuries, illnesses, lost work days, and associated workers' compensation costs. Qualitative evidence from these case studies showed improvements in worker morale, productivity, and product quality.

Psychophysical experiments, explored in Appendix II, (Ex. 27–1) measure subjective responses of individuals performing various laboratory tasks designed to mimic real work procedures. The measures are self-reports of discomfort, fatigue, level of exertion, etc. These measures have been found to correlate well with objective measures of injury frequency in workplaces (Snook, Campanelli, and Hart, 1978, Ex. 26–35; Herrin, Jaraiedi, and Anderson, 1986, Ex. 26–961).

2. NIOSH Summary of the Epidemiological Evidence

The following sections present selected epidemiological evidence organized by risk factor. However, it is helpful first to look at a summary of this evidence, taken from the very thorough analysis carried out by NIOSH (Bernard and Fine, 1997, Ex. 26–1). NIOSH lists reasonable and consistent criteria for including studies in this summary. The Workshop Summary and Papers document from the recent NAS symposium on MSDs (National Academy of Sciences, 1998, Ex. 26–37) contains assessments of the NIOSH analysis by seven respected epidemiologists. This group noted the drawbacks to many of the studies included in the analysis:

- Difficulty in establishing causal direction from any one study.
- Variability in assessment measures (also a strength of the combined body of studies).
- Lack of information concerning disease prevalence in non-working populations.
- The common epidemiological problem of possible unmeasured factors contributing to the effects seen.

However, the group concluded that:

- The NIOSH criteria for study inclusion in the summary were, in general, adequate.
- The preponderance of evidence, particularly from studies with high exposure contrasts among study groups, supports the association between work-related stressors and MSD development.
- The demonstrated reduction of MSDs in workplaces where stressors were reduced also strongly supports this association.

Bernard and contributors (1997, Ex. 26–1) established a four-part classification system to characterize the strength of evidence for work-relatedness, examining the contribution of each risk factor to MSDs, categorized by body location (see Tables V–1 and V–2).

Table V–1.—Upper-Extremity MSDs

MSD LOCATION OR DIAGNOSIS	NUMBER OF STUDIES	RISK FACTOR				
		FORCE	STATIC OR EXTREME POSTURES	REPETITION	VIBRATION (SEGMENTAL)	COMBINATION
Neck and Neck/ Shoulder	>40	++	+++	++	+/0	(—)
Shoulder	>20	+/0	++	++	+/0	(—)
Elbow	>20	++	+/0	+/0	(—)	+++
Carpal Tunnel	>30	++	+/0	++	++	+++
Hand/Wrist Tendinitis	8	++	++	++	(—)	+++
Hand-Arm Vibration	20	(—)	(—)	(—)	+++	(—)

Note: (—) means the association is not reported in the NIOSH publication.

Table V–2.—Lower-Back MSDs

MSD LOCATION OR DIAGNOSIS	NUMBER OF STUDIES	RISK FACTOR				
		HEAVY PHYSICAL WORK	LIFTING AND FORCEFUL MOVEMENTS	STATIC POSTURES	AWKWARD POSTURES	VIBRATION (WHOLE BODY)
Low Back	>40	++	+++	+/0	++	+++

In this determination, the investigators weighted the contribution of individual studies by the quality of the study design:

- Strong evidence of work-relatedness (+++): a very likely causal relationship between exposures of high intensity and/or duration and an MSD, using the epidemiologic criteria for causality (similar to those presented above).
- Evidence of work-relatedness (++): some convincing evidence of a causal relationship.
- Insufficient evidence of work-relatedness (+/0): some suggestion of causality, but most studies lack sufficient quality, consistency, or statistical power; study quality may be lower.
- Evidence of no effect of work factors (-): Adequate studies consistently and strongly show a lack of association between a risk factor and MSDs.

The study considered five categories of risk factors for upper-extremity MSDs (see Table V–1):

- Forceful exertions.
- High levels of static contraction, prolonged static loads or extreme working postures (termed ''awkward postures'' in Section B).
- Highly repetitive work.
- Vibration.
- A combination of these factors.

Table V–1 also shows that there is evidence or strong evidence of work-relatedness for most MSDs and risk factors.

The NIOSH study presents a somewhat different set of risk factors for low-back MSDs (see Table V–2). The classification:

- Looks at static and awkward postures separately, explicitly substituting ''awkward postures'' for extreme postures.
- Inserts ''heavy physical work'' and ''lifting and forceful movements'' in place of ''force.''
- Assesses whole-body vibration instead of segmental vibration.
- Removes assessment of repetition as a separate risk factor.
- Does not address combinations of risk factors.

This modified selection of risk factors is, overall, appropriate to the particular nature of back exposures and injury and reflects the foci of attention in the epidemiological research literature. The last two omissions are unfortunate, however, because both repetition rate and combined exposures to stressors are relevant to the etiology of low-back disorders. In practice, the studies that assessed heavy physical work used definitions of this stressor that include ''high energy demands * * * heavy tiring tasks, manual materials handling tasks, and heavy, dynamic, or intense work'' (Bernard and Fine, 1997, p. 6–4, Ex. 26–1). These stressors probably implicitly include both repetition and a combination of risk factors. Table V–2, like Table V–1, shows that there is evidence or strong evidence of work-relatedness for low back MSDs. Due to the multifactoral nature of MSD causation, the separation of evidence by individual risk factor is artificial. But this separation is useful for clarity and is continued in this section, which presents other epidemiological studies as well as evidence from laboratory and psychophysical studies pointing to the role of workplace stressors in the causation or exacerbation of MSDs.

3. Workplace Risk Factors and MSDs

a. Force.

Epidemiological Evidence. The NIOSH summary (Bernard and Fine, 1997, Ex. 26–1) of upper-extremity MSDs found evidence of a causal relationship between exposure to force and disorders of the neck and elbow, as well as carpal tunnel syndrome (CTS) and hand/wrist tendinitis. (In general, the evidence for work-related MSDs at the elbow has been less convincing than that for other body locations. Although the NIOSH review finds evidence for a relationship between force and epicondylitis, Kourinka and Forcier (1995, Ex. 26–

432) conclude that the evidence is not yet convincing.) Silverstein, Fine, and Armstrong (1987, Ex. 26–34), studying CTS as an outcome, found an OR of 15.5 (95% CI: 1.7–142) for high-force/high-repetition jobs, compared to jobs with low levels of both. The interaction of force and repetition was important in this study; in separate models, force alone had a non-significant OR of 2.9 and repetition alone had an OR of 5.9 ($p < .05$). Nathan *et al.* (1988, Ex. 26–990) also found elevated prevalences of CTS in workers holding high-force/high-repetition jobs. The case definition of these authors did not include self-reported symptoms but only measurable decrements in nerve conduction velocity. This is a stricter case definition than that of Silverstein *et al.* (1987, Ex. 26–34), which was based on self-reported symptoms and physician examinations. This stricter case definition resulted in a smaller but more rigorously defined set of cases; the calculated OR was correspondingly lower (2.0, 95% CI: 1.1–3.4, comparing the highest-force/repetition group to the lowest). Note that this author did not find significant relationships between force and CTS in subsequent work (Nathan *et al.*, 1992, Ex. 26–988).

In addition to the NIOSH summary, other epidemiological studies point to an association between force requirements and work-related MSDs. Silverstein's 1985 cross-sectional research on male and female industrial workers is suggestive, although the NIOSH summary found insufficient evidence for an association between force and shoulder MSDs and did not include this study (Ex. 26–1173). The study compared workers in jobs characterized by a combination of high force and high repetition, measured at the wrist, to those in jobs with low levels of both exposures; the authors calculated an OR of 5.4 (95% CI: 1.3–23) for prevalence of shoulder tendinitis and degenerative joint disease (thus using wrist measurements as a surrogate for shoulder exposure, a possible source of criticism). This study also found an OR for hand/wrist tendinitis of 29 (CI not reported).

Vingärd *et al.* (1991, Ex. 26–1400), in a registry-based cohort study of people hospitalized for osteoarthrosis over 3 years, compared men and women with high exposure to dynamic and static forces at the knee to those with low exposure. Occupations with significantly elevated relative risk were firefighter, farmer, and construction worker for men, and cleaner for women.

Coggon *et al.* (1998, Ex. 26–1285) carried out a case-control study of 611 subjects with hip replacements due to osteoarthritis, compared to matched controls. Men who reported lifting more than 25 kilograms at least 10 times per week for 10 years prior to age 30 or for more than 20 years over their working life had higher rates of surgery (OR 2.7 and 2.3, respectively significant at a 0.05 level). The association did not hold for females.

Laboratory Evidence. Ashton-Miller (1998, Ex. 26–414), summarizing a large body of laboratory evidence assessing the effects of loading on body tissues, concludes that muscle, tendon, and ligamentous tissues can fail when subjected to sufficient force under certain conditions. Faulkner and Brooks (1995, Ex. 26–1410) found that excessive force can cause muscle fiber damage, either by disruption of the actin-myosin (the contractile proteins) interdigitation or of the Z-lines between single sarcomeres (the contractile units in the muscle fibril). Muscles are particularly likely to be injured through exertion of excessive force in eccentric contractions (*i.e.*, as the muscle is being lengthened, such as when stopping the motion of the body or an external object) (Brooks, Zerba, and Faulkner, 1995, Ex. 26–87). Ashton-Miller (1998, Ex. 26–414) suggests that these injuries, although seemingly traumatic, commonly occur in combination with accumulated strain from lower levels of repeated forceful exertions (Wren, Beaupre, and Carter, 1998, Ex. 26–245).

Laboratory evidence for viscoelastic strain in tendons and ligaments under forceful loading is suggestive (*e.g.*, Goldstein *et al.*, 1987, Ex. 26–953; Crisco *et al.*, 1997, Ex. 26–1373). However, more research is necessary to establish whether this strain progresses to MSDs. Animal studies have shown that forceful loading of tendons can produce structural changes similar to those found in MSDs (Rais, 1961, Ex. 26–1166; Backman *et al.*, 1990, Ex. 26–251).

Forceful muscle contraction raises intra-muscular pressure, potentially increasing pressure on nerves and vessels within the active muscle. Abundant animal studies (see summary, Rempel *et al.*, 1998, Ex. 26–444) demonstrate that increased pressure on neurons can reduce blood flow around, and inhibit transport in, axons. Pressure elevations can impair nerve function, increase neural edema, and even alter myelin sheath structure. Many of these changes can occur over relatively short exposure times and in the presence of relatively low pressure elevations. These changes demonstrate a dose-response relationship. This suggests that elevated pressure around nerves during work tasks might cause decrements in nerve function. Both human cadaver studies (Cobb *et al.*, 1996, Ex. 26–98) and work with healthy volunteers (Rempel *et al.*, 1997, Ex. 26–889; Keir *et al.*, 1998, Ex. 26–289) demonstrate that forceful loading of fingertips results in elevated carpal tunnel pressures, well within the range demonstrated to cause damage to animal neurons.

Psychophysical Evidence. Experiments performed over many years at the Liberty Mutual laboratories in Hopkinton, Massachusetts (Snook, 1996, Ex. 26–1353), have examined, in detail, the effects of different biomechanical stressors on subjects' reports of acceptable lifts, carries, pushes, pulls, etc. In general, the experimenter sets all parameters of a simulated task, with the exception of the load, which can be varied by the subject. The subjects are asked to rate task acceptability as if they were performed for a full day, so that the ratings of acceptable load include allowances for fatigue over the course of a workday. The research group has published extensive tables of these acceptable loads (Snook and Ciriello, 1991, Ex. 26–1008). Although there is great individual variation, these experiments generally show the subjects' ability to precisely estimate and regulate the load that would allow them to work a full day without becoming overtired or out of breath. These studies demonstrate the interrelatedness of the biomechanical stressors examined in the Health Effects Section. They show that acceptable load estimates are very sensitive to variations in posture, frequency, and the distance the load is moved.

Klein and Fernandez (1997, Ex. 26–1357) administered a variant of this study design, allowing subjects to adjust the frequency of a repeated pinch grip (determining the maximum acceptable frequency [MAF]) under varying conditions of force, wrist posture, and pinch duration. They found that, as the force of the pinch grip was experimentally increased, the MAF fell.

Summary: Force and Work-Related MSDs. The NIOSH findings of evidence for force-related MSDs in most upper-extremity locations, combined with the few studies addressing lower-extremity MSDs, make a case for a causal association of between increased workplace force requirements and disease. The large number of laboratory studies (see Appendix II, Ex. 27–1) provides evidence for several plausible and repeatable mechanisms by which

forceful exertions could cause MSDs. The psychophysical studies lend support to these conclusions, due to the demonstrated correlation between subjective workload estimates (discomfort, fatigue, and level of exertion) and objectively measured outcomes of injury frequency in workplaces (Snook *et al.,* 1978, Ex. 26–35; Herrin *et al.,* 1986, Ex. 26–961). These studies also demonstrate the interrelatedness of force exposures with several other risk factors for MSDs—in particular, repetition and awkward postures. Taken as a whole, the evidence is consistent and makes a strong case for force as a risk factor for work-related MSDs.

b. Awkward Postures.

Epidemiological Evidence. The NIOSH summary of upper-extremity MSDs (Bernard and Fine, 1997, Ex. 26–1; see Table V–1 above) did not separate static and awkward postures in their conclusions. The summary found evidence of a causal relationship between exposure to static or extreme postures and disorders of the shoulder and hand/wrist tendinitis. There is strong evidence of a causal relationship between postural stressors and neck MSDs. The summary found insufficient evidence for a relationship between these risk factors and elbow disorders or CTS. Of the 15 studies that addressed postures, many with positive results were carried out on VDT workers (*e.g.,* Bernard *et al.,* 1993, Ex. 26–439; Kukkonen *et al.,* 1983, Ex. 26–1138). The research on the largest study population (Linton, 1990, Ex. 26–977) examined combined biomechanical and psychosocial exposures. The study looked at 22,180 Swedish employees undergoing screening examinations at their occupational health care service. Combined exposures to ''uncomfortable posture'' and poor psychosocial work environment showed an OR of 3.5 (95% CI: 2.7–4.5) for neck pain cases (defined as those who reported a visit to a health care professional in the last year for neck pain) compared to low-exposure jobs. The studies in the NIOSH summary support the conclusion that a combination of risk factors carries increased risk. In particular, the studies reviewed provide strong evidence for the causal relationship of combined risk factors (especially force, postural stressors, and repetition) with disorders of the elbow, CTS, and hand/wrist tendinitis.

Other epidemiological studies demonstrate an association between awkward or extreme postures and work-related MSDs. Bjelle *et al.* (1979, Ex. 26–1112) found a strong relationship between industrial work with hands at or above shoulder level and outcomes of shoulder tendinitis (OR: 11; 95% CI: 2.7–42). Similar findings appeared in studies by Herberts *et al.* (Ex. 26–960) on shipyard welders (1981; OR: 13; 95% CI: 1.7–95) and shipyard plate workers (1984; OR: 11; 95% CI: 1.5–83). The referent group in these studies consisted of office workers. A cross-sectional study of female assembly line packers, compared with department store shop assistants (Luopajärvi *et al.,* 1979, Ex. 26–56), found an OR of 7.1 for hand/wrist tendinitis (95% CI: 3.9–12.8). In this study, exposure was a combination of awkward postures, static postures and repetitive motions.

The bulk of the NIOSH-reviewed studies (Bernard and Fine, 1997, Ex. 26–1) do not provide sufficient evidence for the link of postural factors with CTS. However, de Krom *et al.* (1990, Ex. 26–102) found associations between awkward (flexed and extended) wrist postures and CTS. The strength of association increased with hours of exposure. Marras and Schoenmarklin (1993, Ex. 26–172) were able to distinguish between jobs carrying a high and low risk of CTS, using a combination of measured wrist flexion and two dynamic factors (wrist extension angular velocity and wrist flexion angular acceleration).

Laboratory Evidence. Ashton-Miller (1998, Ex. 26–414) cites a number of studies demonstrating that a change of force direction over bony or ligamentous structures creates transverse or shear forces and increases in friction experienced by tendons and tendon sheaths. Increased angles adopted by tendons as they pass around a tendon pulley (related to awkward posture) and increased longitudinal tension (related to the required muscle force) combine to increase friction on the tendon (Uchiyama *et al.,* 1995, Ex. 26–339).

In addition, extreme postures can require elevated muscle activity simply to overcome the resistance of passive tissues. Zipp *et al.* (1983, Ex. 26–1270) found that adopting an extremely pronated forearm position (such as that required by computer keyboard operation) requires high muscle activity, even without any external loading. Non-extreme postures can still trap tissues in injurious positions. Smith, Sonstegard, and Anderson (1977, Ex. 26–1006) demonstrated that even non-extreme wrist flexion can cause the finger flexor tendons to compress the median nerve. Buchholz *et al.* (1988, Ex. 26–1297) detail a sophisticated modeling approach that explains the measured increased muscle force demands associated with non-optimal grip diameters (putting the fingers into awkward biomechanical relationships).

Nerve tissue may also be at risk in anatomical sites associated with awkward posture. Any posture that compresses or crushes a nerve may cause the histological changes noted in Section C.3.a. Human studies (Armstrong *et al.,* 1984, Ex. 26–1293) have shown that histological changes (edema, thickening, fibrosis) occur in nerves at the site of compression injury and possibly at sites of bending (*e.g.,* the ulnar nerve at the elbow). The human cadaver studies (Cobb *et al.,* 1996, Ex. 26–98) and healthy volunteer studies (Rempel *et al.,* 1997, Ex. 26–889; Keir *et al.,* 1998, Ex.26–289) cited above also demonstrate that non-neutral hand postures, combined with forceful loading of fingertips, result in elevated carpal tunnel pressures, well within the range demonstrated to cause damage to animal neurons. Rempel *et al.* (1998, Ex. 26–444) cite eight human studies measuring pressure in the carpal tunnel when the wrist is in a flexed or extended posture relative to a neutral posture. Most of these studies show elevation of carpal pressure, again into the range that causes damage in the animal studies.

Studies of the spine demonstrate similar negative effects of awkward postures. Marras *et al.* (1993, Ex. 26–170) include maximum sagittal trunk flexion angle as one of the five predictors of high risk for low-back injury. In a study by Hutton and Adams (1982, Ex. 26–1381), intervertebral disks in undeviated cadaver spines did not fail until loads exceeded 10,000 Newtons (N). However, disks in extremely flexed spines failed at roughly half that loading (average 5400 N—Adams and Hutton, 1982, Ex. 26–1379). Repetitive loading reduced this average failure point to 3800 N (Adams and Hutton, 1985, Ex. 26–1315). Although the relative magnitude of these forces is important, they may suggest lifting limits that are too high for many living workers. NIOSH, noting the large variability in compression forces associated with disc failure, estimated that 21% of spinal segment specimens would fail at the 3400 N level used as a basis for the NIOSH lifting equation (Waters *et al.,* 1991, Ex. 26–521). Adams *et al.* (1980, Ex. 26–701) report experimental and modeling evidence suggesting that combined forward flexion and lateral bending of the lumbar

spine reduce the injury tolerance of intervertebral disk fibers, possibly increasing chance of rupture. A possible mechanism for disk injury may relate to the fact that lateral flexion and axial rotation of the lumbar spine increase antagonistic muscle activity, thereby increasing the overall disk loading. This is consistent with observations that the combination of lifting, twisting, and bending is one of the most frequent causes of low-back pain (Rowe, 1983, Ex. 26–699).

Psychophysical Evidence. The Liberty Mutual studies cited in Section C.3.a also demonstrate the subjective effect of awkward postures. The maximum acceptable weight (MAW) arrived at by the subjects in these experiments decreased if the lifts were carried out above shoulder height. The MAW was also inversely related to object size (reflecting the fact that moving bulkier loads generally requires more awkward postures).

As described in Section C.3.a, Klein and Fernandez (1997, Ex. 26–1357) allowed subjects adjust to the frequency of a repeated pinch grip (determining the MAF) under varying conditions of force, wrist posture, and pinch duration. They found that the MAF at two-thirds the maximum wrist flexion was significantly less than in a neutral wrist posture. Wrist flexion angle was a significant factor for several variables.

Marley and Fernandez (1995, Ex. 26–863), looking at the stressors associated with hand-held tools, assessed MAF for a simulated drilling task. Compared to ratings in a neutral wrist posture, when the wrist was at one-third maximum flexion, MAF was 88%; at two-thirds maximum flexion, MAF was 73% of the neutral posture value. Subjects used Borg RPE ratings (self-reported ratings of perceived exertion) (Borg, 1982, Ex. 26–705) to estimate required exertion at various body locations. Compared to a neutral wrist position, subjects performing the task with the wrist in two-thirds maximum flexion reported increases in exertion in the wrist, forearm, shoulder, and whole body.

Asymmetrical lifting postures also resulted in a reduction in the MAW. Garg and Badger (1986, Ex. 26–121) asked subjects to carry out a floor-to-table lift twisted 30, 60 and 90 degrees from neutral trunk posture. The MAWs showed significant decreases of 7%, 15%, and 22%, respectively.

Summary: Awkward Postures and Work-Related MSDs. The epidemiological evidence for a causal association between awkward postures and MSDs is strong, especially for neck disorders. Although the NIOSH review (Bernard and Fine, 1997, Ex. 26–1) found insufficient evidence that posture alone can cause CTS, the studies found strong evidence for CTS causation by a combination of risk factors. This suggests that the harmful effects of exposure to awkward posture may be experienced primarily in combination with other risk factors. The numerous laboratory studies examining the relationship between postural stressors and CTS, in particular, strengthen the evidence for a combination of awkward postures and force as risk factors for this outcome. Likewise, extensive epidemiological and laboratory evidence for increased risk of low-back injury due to bending and twisting also demonstrates the important role that postural stressors play in MSD causation.

This evidence is further strengthened by the sensitivity to postural variables of subject-estimated safe loads in the psychophysical literature. These psychophysical studies lend support to these conclusions, due to the demonstrated correlation between subjective workload estimates (discomfort, fatigue and level of exertion) and objectively measured outcomes of injury frequency in workplaces (Snook *et al.*, 1978, Ex. 26–35; Herrin *et al.*, 1986, Ex. 26–961). These studies also demonstrate the interrelatedness of postural exposures with several other risk factors for musculoskeletal disorders, in particular, repetition and force. The convergent evidence from these diverse areas, with very different methodological approaches, strongly supports the hypothesis that awkward postures have a causal role in the etiology of MSDs.

c. Static Postures.

Epidemiological Evidence. Since the NIOSH summary (Bernard and Fine, 1997, Ex. 26–1) did not distinguish between awkward and static postures, the summary in section C.3.b applies here as well. In addition to the NIOSH summary (see Tables V–1 and V–2 above), other epidemiological studies demonstrate an association between static contractions or prolonged static load and work-related MSDs. In a review of the epidemiological evidence for three neck-related MSDs, the contributors to Kourinka and Forcier (1995, Ex. 26–432) report consistent associations between exposures to static head and arm postures and outcomes of tension neck syndrome. Grieco *et al.* (1998, Ex. 26–627) also report associations between static work and tension neck syndrome in several different occupations. Looking at the neck region more generally, Hales and Bernard (1996, Ex. 26–896) report several studies showing consistent association between neck disorders and work involving static or constrained postures. A review of neck studies by Hidalgo *et al.* (1992, Ex. 26–631) proposes that prolonged static contraction of neck muscles be limited to force levels at or below 1% of maximum voluntary contraction (MVC). In an intervention study, Aarås *et al.* (1998, Ex. 26–597) found that introduction of a workstation arrangement that allowed forearm support (thus lowering static load on the shoulders) reduced trapezius muscle activity from 1.5% to 0.3% of MVC and was associated with a reduction in neck pain.

A cross-sectional study of 152 female assembly line packers, compared with department store shop assistants (Luopajärvi *et al.*, 1979, Ex. 26–56), found an OR of 7.1 for hand/wrist tendinitis (95% CI: 3.9–12.8). In this study, exposure was a combination of static postures, awkward postures and repetitive motions.

A population-based case-control study (Cooper *et al.*, 1994, Ex. 26–460), comparing cases with knee osteoarthritis to matched controls with non-arthritic knee pain, found that squatting more than 30 minutes per day was associated with an increased prevalence of osteoarthritis (OR: 6.9, 95% CI 1.8—26.4). Vingärd *et al.* (1991), in a registry-based cohort study of people hospitalized for osteoarthrosis over 3 years, compared men and women with high exposure to static and dynamic forces at the knee to those with low exposure. Occupations with significantly elevated relative risk were firefighter, farmer, and construction worker for men, and cleaner for women.

Laboratory Evidence. In general, the laboratory literature cited above for force and awkward posture is relevant to the prolonged exposures involved in static postures (Zipp *et al.*, 1983, Ex. 26–1270; Buchholz *et al.*, 1988, Ex. 26–1297; Smith, Sonstegard, and Anderson, 1977, Ex. 26–1006). Many of the same mechanisms apply, but the duration is increased and the temporal profile of exposure is made worse by the reduction in rest breaks and opportunity for recovery time. Lundborg *et al.* (1982, Ex. 26–979) showed that a constant hydrostatic pressure (*i.e.*, during a static muscle contraction) of between 30 and 60 mm Hg reduces microcirculation of the nerve and compromises nerve conduction.

Rohmert (1973, Ex. 26–580) found that muscle contractions can be maintained for prolonged periods if kept below 20% of MVC. But other investigators (Westgaard and Aarås, 1984, Ex. 26–1026) found chronic deleterious effects of contractions even if they are lower than 5% of MVC. This latter finding is supported by the observation that low-level static loading (such as shoulder loading in keyboard tasks) is associated with shoulder MSDs (Aarås *et al.,* 1998, Ex. 26–597). The supraspinatus muscle, a muscle severely constrained by bone and ligamentous tissue, demonstrates increased intramuscular pressure during small amounts of shoulder abduction or flexion (Järvholm *et al.,* 1990, Ex. 26–285). This suggests the possibility of chronic blood vessel and nerve compression during static tasks.

Chronic reduction of blood flow may be a mechanism by which static muscle contractions lead to MSDs. Several studies have found that the small, slow motor units in patients with chronic muscle pain show changes consistent with reduced local oxygen concentrations (Larsson *et al.,* 1988, Ex. 26–1140; Dennett and Fry, 1988, Ex. 26–104). Reduced blood flow and disruption of the transportation of nutrients and oxygen can produce intramuscular edema (Sjogaard, 1988, Ex. 26–206). The effect can be compounded in situations where recovery time between static contractions is insufficient. Eventually, a number of changes can result: muscle membrane damage, abnormal calcium homeostasis, an increase in free radicals, a rise in other inflammatory mediators, and degenerative changes (Sjogaard and Sjogaard, 1998, Ex. 26–1322).

Psychophysical Evidence. Several studies have evaluated the maximum acceptable weight (MAW) in conditions requiring prolonged stooping (low ceiling height). Smith *et al.* (1992, Ex. 26–1007) performed laboratory experiments on 100 subjects (50 male, 50 female) recruited from a college-age population at Texas Tech University. The study collected data on a number of awkward postures, such as twisting, lying down, kneeling, squatting, and carrying loads with a restricted ceiling. The authors found that the MAW decreased with decreasing ceiling height (which requires forward flexion during lifting) as well as with twisted postures.

Klein and Fernandez (1997, Ex. 26–1357) allowed subjects to adjust the frequency of a repeated pinch grip (determining the MAF) under varying conditions of force, wrist posture and pinch duration. They found that, as the pinch grip was held for longer increments of time (1, 3, and 7 seconds), the MAF fell.

Summary: Static Postures and Work-Related MSDs. The epidemiological evidence is particularly strong for the causal role of static postures in MSDs of the neck and shoulder region. This evidence is suggestive but less convincing for disorders of the distal upper extremities. Laboratory evidence for muscle and tendon damage in these areas, as well as secondary compression of blood vessels and nerves, lends support to the connection between work-related static postural requirements and the development of these disorders. The psychophysical studies have not generally focused on static postures, but the two studies cited in section C.3.c provide evidence of increased fatigue and discomfort related to static postures of the back and fingers. These psychophysical studies lend support to the conclusions of work-relatedness, due to the demonstrated correlation between subjective workload estimates (discomfort, fatigue, and level of exertion) and objectively measured outcomes of injury frequency in workplaces (Snook *et al.,* 1978, Ex. 26–35; Herrin *et al.,* 1986, Ex. 26–961). These studies also demonstrate the interrelatedness of postural exposures with several other risk factors for musculoskeletal disorders, in particular repetition and force. Taken as a whole, the evidence suggests that static postures are causal factors in the etiology of MSDs, both through exacerbation of the mechanisms explored under other risk factors (*e.g.,* awkward postures, force) and through chronic reductions in blood flow and neural function caused by prolonged elevations of intramuscular pressure.

d. Repetition. Repetition has qualities of both a risk factor and a modifying factor (or ''characteristic property'' (ANSI, 1998, Ex. 26–1264)). Because of this borderline position, repetition is often reported as an exposure intensifier (*e.g.,* Radwin and Lavender, 1998, Ex. 26–37) and often as a risk factor in itself (*e.g.,* Kourinka and Forcier, 1995, Ex. 26–432). Thus, a substantial portion of the evidence presented in subsequent sections, supporting the association of repetition with work-related MSDs, examines repetition in combination with other risk factors. In fact, the NIOSH summary (Bernard and Fine, 1997, Ex. 26–1) found that a combination of risk factors increases the strength of the evidence for work-relatedness. This suggests that each individual risk factor has characteristics of both a basic risk factor and a modifier, and the distinction becomes somewhat academic.

Epidemiological Evidence. The NIOSH summary (Bernard and Fine, 1997, Ex. 26–1; see Table V–1 above) found evidence for work-related MSDs connected with exposure to repetitive work for all body locations considered except the elbow. Of the 16 selected studies that addressed repetition exposure and found a positive association with neck disorders, 11 found associations that were statistically significant. Ohlsson *et al.* (1995, Ex. 26–868) compared 82 female industrial workers exposed to short-cycle tasks (less than 30 seconds) to 64 referents with no exposure to repetitive work. The OR for tension neck syndrome was 3.6 (95% CI: 1.5–8.8), and the OR for shoulder symptoms (several types of tendinitis, frozen shoulder, acromioclavicular syndrome) was 5.0 (95% CI: 2.2–11.0). Silverstein *et al.* (1987, Ex. 26–34), studying CTS as an outcome, found an OR of 15.5 (95% CI: 1.7–142) for high-force/high-repetition jobs, compared to jobs with low levels of both. Jobs with only high-repetition exposure still demonstrated an OR of 5.5, compared to low-force/low-repetition jobs. Nathan *et al.* (1988, Ex. 26–990) also found an elevated prevalence of CTS in workers holding high-force/high-repetition jobs. Their stricter case definition was based on nerve conduction velocity decrements, and the calculated OR was correspondingly lower (2.0, 95% CI: 1.1–3.4). Note that subsequent investigations by this investigator did not find a significant association of repetition with CTS (Nathan *et al.,* 1992, Ex. 26–988).

Other epidemiological studies demonstrate an association between repetitive movements and work-related MSDs. The contributors to Kourinka and Forcier (1995, Ex. 26–432), in a review of the epidemiological evidence for three neck-related MSDs, report weak-to-moderate, but consistent associations between exposures to repetitive work and outcomes of tension neck syndrome and thoracic outlet syndrome (TOS). They and other reviewers (*e.g.,* Grieco *et al.,* 1998, Ex. 26–627) did not find convincing evidence of a connection between repetition and cervical radiculopathy. Looking at the neck region more generally, Hales and Bernard (1996, Ex. 26–896) report several studies showing consistent association between neck disorders and repetitive work/forceful repetitive work.

Silverstein's (1985, Ex. 26–1173) cross-sectional study of male and female industrial workers compared workers in

jobs characterized by a combination of high force and high repetition to those in jobs with low levels of both exposures. She calculated a risk ratio of 5.4 (95% CI: 1.3–23) for prevalence of shoulder tendinitis and degenerative joint disease. This study found an OR for hand/wrist tendinitis of 29 (CI not reported). A cross-sectional study of female assembly line packers, compared with department store shop assistants (Luopajärvi *et al.,* 1979, Ex. 26–56), found an OR of 7.1 for hand/wrist tendinitis (95% CI: 3.9–12.8). In this study, exposure was a combination of awkward postures, static postures and repetitive motions. Other studies have also demonstrated a strong association between CTS and repetition (reviewed in Kourinka and Forcier, 1995, Ex. 26–432).

A population-based case-control study (Cooper *et al.,* 1994, Ex. 26–460), comparing cases with knee osteoarthritis to matched controls with non-arthritic knee pain, found that climbing more than 10 flights of stairs per day was associated with increased prevalence of osteoarthritis (OR: 2.7, 95% CI 1.2–6.1).

Laboratory Evidence. In 1951, Sperling (Ex.26–1411) subjected his own fingers to a series of prolonged, repetitive movements, against resistance. In all cases, the area around the affected tendon became tender and swollen, and in most cases, he began to notice snapping and thickening. These symptoms remained for several months. Sperling concluded that tendon injury could be caused by simple, repetitive loading, without the necessity for traumatic injury. Rais (1961, Ex. 26-1166) performed two experiments subjecting rabbits to varying degrees of stressful, repetitive leg movement. Overall, he found evidence of peritendinitis, localized to the area of the myotendinous junction. The changes indicated cellular damage and restorative activities. In the muscles themselves, he also observed degeneration of varying degrees, fibrin deposition, and evidence of regeneration.

Experimentally, Hagberg (1981, Ex. 26–955) demonstrated that a 1-hour course of repetitive shoulder flexion movements could induce acute shoulder tendinitis. Several investigators found an increase in shoulder muscle activity and/or pain when assembly line work pace was increased (*e.g.,* Odenrick *et al.,* 1988, Ex. 26–576; Ohlsson *et al.,* 1989, Ex. 26–1290). These findings should be interpreted with caution: Shoulder tension is strongly affected by psychosocial factors (although it should be noted that the overall effect is still the increase of shoulder muscle activity).

A few investigators have studied the effects of repeated loading on cadaver spinal segments (Brinckmann, *et al.,* 1987, Ex. 26–1318; 1988; Hansson, *et al.,* 1987, Ex. 26–279). These studies applied a submaximal load (a percentage of the load associated with failure in a single application). A strong dose-response relationship emerged. Even with compressive loads set at 55% of the single trial failure load, mechanical failure occurred in 92% of the specimens after 5000 cycles. At 65% of this load, 91% of the specimens failed after only 500 cycles. At 75% of this load, some specimens failed after only 10 cycles. Although cadaver tissue probably acts differently from living tissue, these results do suggest that repetition is a risk factor for spinal injury.

Psychophysical Evidence. The Liberty Mutual studies cited in Section C.3.a.iii also demonstrate the subjective effect of repetition rates on subject estimates of tasks that could be performed over the course of a work day without undue fatigue, discomfort, or overexertion (Snook, 1996, Ex. 26–1353). As noted above, the experimenter sets all parameters of a simulated task, with the exception of the load, which can be varied by the subject. The subjects are asked to rate task acceptability as if they were performing the task for a full workday, so the ratings of acceptable load include allowances for fatigue over the course of a workday. The research group has published extensive tables of these acceptable loads (Snook and Ciriello, 1991, Ex. 26–1008). Although there is great individual variation, these experiments in general show the subjects' ability to precisely estimate and regulate the load that would allow a full day of work without becoming overtired or out of breath. These studies show that acceptable load estimates are very sensitive to variations in the repetition rate of the task. In all variations, the MAW that was estimated by the subjects in these experiments decreased as the frequency of the lift, lower, push, or pull increased.

Separate studies by Garg and Banaag (1988, Ex. 26–951) and Mital and Fard (1986, Ex. 26–182), in addition to replicating the MAW decrements attributable to asymmetric lifting noted under ''awkward postures,'' also found that increased frequency of lifting reduced the MAW reported by their subjects. Klein and Fernandez (1997, Ex. 26–1357) administered a variant of this study design, allowing subjects to adjust the frequency of a repeated pinch grip (determining the MAF) under varying conditions of force, wrist posture, and pinch duration. They found that, as force of the pinch grip was experimentally increased, the MAF fell.

Summary: Repetition and Work-Related MSDs. Despite the difficulties in assessing repetition in isolation from other risk factors, the epidemiological evidence strongly implicates repetitive motions in the etiology of work-related MSDs. A large body of laboratory studies demonstrates a biological plausibility for this relationship. The psychophysical research lends support to the epidemiological and laboratory results: it demonstrates a correlation between subjective workload estimates (discomfort, fatigue, and level of exertion) and objectively measured outcomes of injury frequency in workplaces (Snook *et al.,* 1978, Ex. 26–35; Herrin *et al.,* 1986, Ex. 26–961). These studies also demonstrate the interrelatedness of repetition with several other risk factors for musculoskeletal disorders, in particular, force and awkward postures. In sum, the congruence of evidence from several different research traditions, with different methodologies, strongly implicates repetition in the etiology of work-related MSDs.

e. Dynamic Factors.

Epidemiological Evidence. The contributors to the NIOSH summary (Bernard and Fine, 1997, Ex. 26–1) did not examine evidence linking dynamic factors with work-related MSDs. Most research on dynamic factors has been carried out on low-back injury. Sudden maximal lifting effort and unguarded movements appear to be risks for developing work-related low-back pain (Magora and Schwartz, 1976, Ex. 26–389). Marras and Granata (1995, Ex. 26–1383) categorized jobs into three levels of risk (meaning risk of low-back injury, assessed by medical reports). They then calculated ORs of a job, characterized by five measures of exposure falling into the high-risk category. The OR of a job with the highest combined exposure score, compared to the lowest combined score, was 10.7 (95% CI: 4.9–23.6). These exposure measures (assessed by sophisticated electrogoniometry) include dynamic factors: linear and angular velocity and acceleration of the lumbar spine. Marras and Schoenmarklin (1993, Ex. 26–172) also implicate dynamic factors in wrist MSDs. Using a similar, job-based analytic design, they found that angular velocity of wrist

extension and angular acceleration of wrist flexion could distinguish between jobs having high and low prevalence of CTS.

Laboratory Evidence. The most persuasive evidence for the risks associated with dynamic factors comes from work on the intervertebral disks. Marras and Granata demonstrated that the magnitude of compressive and shear forces on the disks is related to the speed and acceleration of movement in both lateral bending (1997, Ex. 26–169) and twisting (1995, Ex. 26–1383). Degree of asymmetry also affects the trunk motion characteristics associated with increased risk of back injury (Marras *et al.*, 1993, Ex. 26–170). Velocity and acceleration measures were all higher with one-handed lifts, the size of increase being proportional to the angle of asymmetry.

Szabo and Chidgey (1989, Ex. 26–1168) found that repetitive, passive wrist flexion and extension resulted in higher pressures in the carpal tunnel. These elevated pressures took longer to return to normal in their CTS patients than in normal subjects. These investigators also found evidence that, if the wrist and finger motions are active (in other words, if the subject rather than the investigator moves the wrist), the effect may be larger.

Psychophysical Evidence. The psychophysical laboratory studies have not explicitly examined the impact of dynamic factors, although it is likely that the studies of repetition (Section C.3.d) do address dynamic factors by proxy (Snook, 1996, Ex. 26–1353; Snook and Ciriello, 1991, Ex. 26–1008; Garg and Banaag, 1988, Ex. 26–951; Mital and Fard, 1986, Ex. 26–182; Klein and Fernandez, 1997, Ex. 26–1357). Increased repetition rates necessarily entail increases in angular and linear velocity and acceleration of some body segments. The resultant increases in forces experienced by body tissues (*e.g.*, Marras and Granata, 1995, 1997 Exs. 26–1383 and 26–169) might explain the subjective perceptions of fatigue and discomfort that result in a particular estimated MAW.

Summary: Dynamic Factors and Work-Related MSDs. Attention to dynamic factors in their own right (as opposed to the proxy representation of repetition) is very recent. The bodies of epidemiological and laboratory evidence relating dynamic stressors to MSD development are consistent with each other and with research centered on the other risk factors. But the existing studies are limited in number and in scope. As a result, the literature does not allow quite as much confidence in connecting these factors with work-related MSDs as can be demonstrated for the other risk factors addressed in this section. Further research is needed to more firmly establish the link between dynamic factors and work-related MSDs.

f. Compression. The classification of risk factors presented in Section B separated compression into external and internal compression. Internal compression has been addressed above, as the consequence of other biomechanical exposures, such as force, awkward and static postures, and repetition. This section only addresses externally applied compressive forces.

Epidemiological Evidence. The NIOSH summary (Bernard and Fine, 1997, Ex. 26–1) did not examine the association of compressive forces with MSDs. A few epidemiological studies have assessed the role of compression as a risk factor. Hypothenar hammer syndrome, characterized by signs of blood deprivation in the fingers, is caused by thrombosis or aneurysm in the ulnar artery or the superficial palmar arterial arch. This condition has been linked to the practice of using the palm as a hammer, exposing the palm to repetitive, forceful compression. Little and Ferguson (1972, Ex. 26–1144) calculated an OR of 16.3 (95% CI: 2.7–100) for objectively verified (by a Doppler flow detector) ulnar artery block, comparing vehicle maintenance workers who used their hands as a hammer (n=79) to those who did not (n=48). Nilsson *et al.* (1989, Ex. 26–1148) found a smaller effect (OR: 2.8; 95% CI: 1.3–6.2), comparing 890 plate workers to 61 office workers in the same plant. This study also found a dose-response relationship, with the OR increasing with increasing years on the job. However, inappropriate palm use and vibration exposure occurred together in this population.

Two studies also link bursitis of the knee with jobs that require a substantial amount of time in a kneeling position. Thun *et al.* (1987, Ex. 26–60) found a non-significant prevalence ratio for bursitis of 3.2 (90% CI: 0.8–3.9), comparing tile and terrazzo setters to bricklayers and millwrights. Kivimäki *et al.* (1992, Ex. 26–1137), comparing carpet layers to painters, calculated an OR of 11.2 (95% CI: 3.4–38) for doctor-diagnosed prepatellar bursitis. A population-based case-control study (Cooper *et al.*, 1994, Ex. 26–460) compared cases with knee osteoarthritis to matched controls with non-arthritic knee pain. They found that kneeling more than 30 minutes per day was associated with increased prevalence of osteoarthritis (OR: 3.4; 95% CI: 1.3–9.1).

Laboratory Evidence. Most of the research concerning the relationship of mechanical compression to MSDs has been conducted in the laboratory. Researchers have known for years that tools with inappropriately short handles, such as pliers and paint scrapers, can apply substantial compressive force to the blood vessels and nerves in the palmar area, resulting in occlusion of the ulnar artery, in particular, and possible neuropathy (Tichauer; 1966, Ex. 26–1172; Tichauer and Gage, 1977, Ex. 26–1269). There is medical evidence for compression-related MSDs. Finelli (1975, Ex. 26–115) describes the compression of an ulnar nerve branch in the palm by both occupational (tool handles) and non-occupational (bicycle handle grips) exposures. Sauter *et al.* (1987, Ex. 26–199) present a case example of injury due to wrist compression at a keyboard job. Several investigators describe compression of the ulnar nerve at the elbow, caused by leaning the ulnar side of the elbow on a hard surface (*e.g.*, Aguayo, 1975, Ex. 26–702). Nevasier (1980, Ex. 26–394) found examples of shoulder tenosynovitis in individuals who habitually carried heavy loads (such as lumber) on their shoulder.

Psychophysical Evidence. Psychophysical studies have not examined the effects of compression.

Summary: Compression and Work-Related MSDs. Despite the long history of recognition (particularly the relationship between tool handles and palmar compression), relatively little research has been performed on this risk factor. The existing epidemiological and laboratory evidence is congruent in suggesting the linkage between compression and at least two medical conditions. Particularly in the case of hypothenar hammer syndrome, a plausible physiologic mechanism exists.

g. Vibration.

Epidemiological Evidence. The NIOSH summary (Bernard and Fine, 1997, Ex. 26–1; see Table V–1 above) finds strong evidence for a causal relationship between segmental vibration and hand-arm vibration syndrome (HAVS). The only study to meet all four of the NIOSH inclusion criteria (Bovenzi *et al.*, 1995, Ex. 26–354) compared forestry workers with more than 400 hours of sawing to shipyard workers

with no vibration exposure. These authors found increasing effect sizes, depending on the intensity of vibration exposure. The OR for forestry workers using anti-vibration saws was 6.2 (95% CI: 2.3–17.1); the OR for workers using no anti-vibration measures was 32.3 (95% CI: 11.2–93). This study also found a dose-response relationship to number of years exposed. Nilsson *et al.* (1989, Ex. 26–1148), comparing platers with current vibration exposure to office workers in the same workplace, calculated an OR of 85 (95% CI: 15–486). The high ORs in these studies have large confidence intervals but demonstrate the strength of effect that is characteristic of many vibration studies.

Other epidemiological studies demonstrate an association between vibration and work-related MSDs. Most work reported in the Health Effects Section addresses segmental vibration exposure of HAVS or occupational Raynaud's syndrome. Studies of select populations using vibrating tools find high concentrations of vascular and neurological symptoms compared to these in other working populations. Examples include shipyard workers (Cherniack *et al.,* 1990, Ex. 26–1116), surgeons (Cherniack and Mohr, 1994, Ex. 26–1341), and dental technicians (Hjortsburg, 1989, Ex. 26–1131).

The NIOSH summary also found evidence for a causal link between segmental vibration and CTS. Chatterjee *et al.* (1982, Ex. 26–941) compared 16 rock drillers to 15 controls unexposed to vibration. The OR for CTS, identified by nerve conduction studies, was 10.9 (95% CI: 1.02–524). Weislander *et al.* (1989, Ex. 26–1027), comparing 32 male CTS patients to population referents, found an OR for vibrating tool use of 6.1 (95% CI: 2.4–15). Several other studies have also found an association between CTS and vibration exposure in jobs involving the use of vibrating tools, such as grinders and chipping hammers (*e.g.,* Nathan *et al.,* 1988, Ex. 26–990; Hagberg *et al.,* 1992, Ex. 8–1). In this literature, however, it is extremely difficult to separate the association of CTS and vibration from the association of CTS and the other biomechanical stressors that often are associated with these tools: awkward and static postures, repetition, and high force requirements.

Some literature has addressed the consequences to other body parts of whole-body vibration exposure to other body parts. Hedlund (1989, Ex. 26–1279) found a foot analogue of HAVS in miners exposed to whole-body and segmental vibration. However, other research suggests that foot symptoms may be a more generalized sympathetic nervous system response to segmental exposure in the upper extremities (Sakakibara *et al.,* 1991, Ex. 26–1356). Other studies of whole-body vibration have suggested links to driving. Jensen *et al.* (1996, Ex. 26–145), studying a cohort of more than 89,000 drivers hospitalized for prolapsed cervical disks over 10 years, found a Standardized Hospitalization Ratio (SHR) of 142 (95% CI: 126.8–159.6), compared to other male workers. They also reported a prevalence ratio for self-reported vibration exposure of 7.1 (95% CI: 4.1–11.7) for the drivers. This research did not directly link vibration exposure with outcomes of prolapsed cervical disk.

Laboratory Evidence. Short-term and long-term changes to human neural tissue have been demonstrated by a number of researchers. These effects include intraneural edema, structural changes in non-myelinated fibers, demyelination, fibrosis, and even loss of axons (Takeuchi *et al.,* 1988, Ex. 26–682; Stromberg *et al.,* 1997, Ex. 26–894). Chang *et al.* (1994, Ex. 26–357) found similar changes in rat peripheral nerves. Finger biopsies of workers heavily exposed to local vibration have shown signs of significant endothelial injury (Takeuchi *et al.,* 1986, Ex. 26–681).

In the back, vibration may diminish the blood flow to the intervertebral disks. This has been demonstrated by Hirano, Tsuji, and Oshima (1988, Ex. 26–140) in rabbit intervertebral disks exposed to in vivo vibration. This could predispose the spine to injury by reducing both the transport of nutrients to the disk interior and the degree of hydration necessary to support the spine under load.

Psychophysical Studies. Although the weighting curves established for vibration exposure rely heavily on perceived discomfort, no formal psychophysical laboratory work has been performed on vibration.

Summary: Vibration and Work-Related MSDs. Vibration is the one biomechanical stressor that may be able to cause a specific disease (HAVS) as the only exposure. The epidemiological evidence is considered strong for vibration as the only causal factor for this outcome. Epidemiological evidence also exists for a causal link between vibration exposure and CTS.

The laboratory evidence supports these conclusions with findings of anatomical and physiological changes, due to segmental vibration, that are consistent with the symptoms and signs of HAVS. This congruent evidence strongly supports the implication of segmental vibration as the risk factor for the development of HAVS.

The evidence supporting the association between whole-body vibration exposure and disk degeneration is not as strong, but it is suggestive. More research into this association is required.

4. Modifying Factors and MSDs

Many of the studies cited above also indicate the importance of the modifying factors in this section's classification scheme: intensity/magnitude, duration, temporal profiles, and cold temperatures. Much of the research summarized by Bernard and Fine (1997, Ex. 26–1) finds that exposures characterized by high intensity and/or duration are associated with higher levels of MSD outcome than those with lower levels of these modifiers. These two modifiers are examined more fully in Section C.5, below.

a. Intensity. Intensity is included in many of the epidemiological and laboratory studies cited above. In particular, studies assessing the effects of high and low force are based in measures of intensity. The evidence for intensity as an important modifier of exposure in MSD etiology is presented below, in Section C.5.

b. Duration. As with intensity, duration is often the measure of high and low exposure in studies cited above. Much epidemiologic research measures the hours of exposure and has documented a dose-response relationship between duration and MSD outcomes. For example, Brisson *et al.* (1989, Ex. 26–937) found that the length of exposure to piecework in the garment industry was associated with increased MSD levels. de Krom *et al.* (1990, Ex. 26–102) found that hours of exposure increased the association of awkward, flexed wrist postures with CTS. Hagberg *et al.* (1990, Ex. 26–1317) demonstrated a duration/MSD association for vibration exposure. Kourinka and Forcier (1995, Ex. 26–432) summarize a collection of similar studies, all of which find that length of exposure, either per day or over a lifetime, increases the size of the association between exposure and work-related MSD outcome.

Duration may be measured in much longer time spans than hours. Anderson and Felson (1988, Ex. 26–926), analyzing the First National Health and Nutrition

Examination Survey (HANES I) data, found that an increased risk of osteoarthritis related to job characteristics appeared only in older workers, suggesting that lifelong exposure may be a part of the etiology.

The evidence linking duration with MSD causation is presented in detail below, in Section C.5.

c. Temporal Profile (Fatigue/Inadequate Recovery Time). In general, repeated damage to body tissues without adequate recovery time for repair may create permanent structural damage. Fatigue has been shown to modify muscle response to external load. As noted above, when muscles fatigue, the characteristics and effects of internal muscle loading can be changed in two ways. Within a given muscle, fiber recruitment generally proceeds from small to large fibers. Some small, slow-twitch fibers may be almost constantly in use and become fatigued and possibly injured, even during very-low-force contractions (see Section C.3.c) (Radwin and Lavender, in NAS, 1998, Ex. 26–37). This phenomenon, termed the ''Cinderella fiber theory,'' is discussed in more detail in later sections. This theory suggests one physiological reason that adequate rest cycles in work activities are important.

d. Cold Temperatures. Research has strongly linked cold to the exacerbation of effects due to vibration exposure. Lundström and Johansson (1986, Ex. 26–164) demonstrated the reduction in mechanoreceptor sensitivity with combined exposure to vibration and cold. This was accompanied by an increase in finger force exerted by subjects, creating better coupling between hand and vibration source and increasing the amount of vibration absorbed by the upper extremities. Simultaneously, this increased force is itself a possible risk factor for CTS.

Cold temperatures may also increase muscle activation required for a given task. Hammerskjöld *et al.* (1992, Ex. 26–957) found increased EMG signals in carpenters after hand exposure to cold, as well as increased perceived exertion and increased time required to carry out nailing tasks. Riley *et al.* (1983, Ex. 26–1358) showed that exposure to cold temperatures resulted in decreased performance on an assembly task. The experimentally demonstrated decrease in strength and coordination of the hands after exposure to cold (*e.g.*, Vangaard, 1975, Ex. 26–506; Vincent and Tipton, 1988, Ex. 26–592) may be the mechanism through which greater force requirements are made on muscles and tendons, causing or exacerbating MSDs.

e. Summary: Modifiers and Work-Related MSDs. The evidence for the effects of these modifying factors is contained within each risk factor section, as well as in the brief review above. Section C.5 below explores the evidence for the roles of intensity and duration in modifying the relationship of stressors to MSD outcomes. This evidence makes a strong case for the impact that each of these workplace modifiers has on the way the body tissues receive a given ''dose'' of a biomechanical stressor and the way in which that tissue can process, repair, and recover from this dose.

5. Evidence for the Relationships Between Exposure Intensity and MSD Prevalence

This section reviews studies designed to examine the relationships between intensity and/or duration of exposure to workplace risk factors and the magnitude of the risk for developing a work-related MSD (typically measured as an OR). In this capacity, the section reviews some of the studies presented above in greater detail. Data demonstrating a positive relationship between exposure and response provide evidence for a causal relationship between exposure to the hazard in the workplace and an increase in the occurrence and/or severity of the adverse response. Often, regression analysis is used to verify that the relationship is statistically significant even when potential confounding factors, such as gender and age, are taken into consideration. The strength of the association between exposure and response is reflected in the slope of the exposure-response curve; as the slope increases, the strength of the association increases and provides greater evidence of a causal relationship between exposure to the hazard of interest and increased risk of injury or illness.

Generalized models do not exist that would permit OSHA to use these data to quantify risk across all working populations. Nevertheless, these studies are useful to illustrate the extent to which risk can be reduced by reducing the intensity and duration of exposures to workplace risk factors.

The relationship between duration of exposure to workplace risk factors and prevalence of MSDs has been demonstrated in numerous studies. For example, the 1988 Occupational Health Supplement to the National Health Interview Survey (NHIS–OHS) conducted by the National Center for Health Statistics (NCHS) showed a clear dose-response relationship between hours engaged in manual handling and episodes of back pain lasting 7 days or longer. NCHS interviewed 27,408 currently employed workers between 18 and 64 years of age to gather information on the health conditions of the currently employed noninstitutionalized civilian population and to develop weighted national estimates of the incidence of health conditions, including episodes of back pain, known to occur in association with employment. All estimates were based on self-reports.

NIOSH (Exs. 26–1104, 26–1105, 26–1106) used the NCHS data to develop weighted national estimates of the number of currently employed workers by the status of back pain episodes lasting 1 week or longer, and by number of hours exposed to some of the workplace risk factors associated with MSDs of the back: strenuous physical activity and repeated bending, twisting, or reaching. Exposure was divided into categories of 0 hours, 0 to less than 2 hours, 2 to less than 4 hours, 4 to less than 6 hours, 6 to less than 8 hours, and 8 hours or more.

Of particular interest were:

- The number of currently employed workers experiencing no episodes of back pain.
- The number of currently employed workers experiencing an episode of back pain lasting 1 week or longer due to repeated activities at their current or most recent job and not due to any accident.

With these data categorized by hours of exposure to workplace risk factors, ORs could be calculated for episodes of back pain due to repeated activities at work for each of the exposure categories and each of the workplace risk factors considered.

Table V–3 presents the estimated number of currently employed workers engaged in strenuous physical activity such as lifting, pushing, or pulling heavy objects. Table V–4 presents the estimated number of currently employed workers engaged in repeated bending, twisting, or reaching. In each table the estimated numbers are broken down by hours per day engaged in each of the work activities, and by back pain status (either none or an episode lasting at least 1 week due to repeated activities at a current or most recent job and not due to any accident). In addition, ORs are presented.

The ORs in Table V–3 clearly indicate that exposure to strenuous physical activity increases the risk of episodes of back pain. The data show a clear positive exposure-response trend: the risk of episodes of back pain increases with an increase in the daily number of hours engaged in strenuous physical activity. Table V–4 shows the same results: the risk of episodes of back pain increases as the number of hours engaged in repeated bending, twisting, or reaching increases. These results are shown graphically in Figure V–1. They indicate that the risk of severe back pain can be reduced substantially by reducing the daily duration of exposure to these risk factors. For example, the risk can be reduced by about half if exposure to these risk factors is reduced from 6 to 8 hours to 2 hours or less per day.

Table V–3 shows that for some exposure categories, the ORs do not increase as exposure increases. The OR for workers engaged in strenuous physical activity for 6 to 8 hours is lower than the OR for workers engaged in strenuous physical activity for 4 to 6 hours. This deviation from an increasing trend, however, does not mean that there is no such trend. NIOSH used its estimated numbers to conduct a logistic regression of episodes of back pain on duration of exposure, adjusting for age and gender. The parameter estimates for each of the two types of exposure were positive and highly statistically significant ($p < .01$). This means that the increasing trend observed in the relationships between episodes of back pain and duration of each type of exposure is statistically significant.

Table V–3.—Estimated Number of Currently Employed Workers Engaged in Strenuous Physical Activity Such as Lifting, Pushing, or Pulling Heavy Objects, by Duration and Back Pain Status [1]

HOURS ENGAGED	BACK PAIN				PERCENT	ODDS RATIO [4]
	NONE		AT LEAST 1 WEEK DUE TO REPEATED ACTIVITIES AT WORK [3]			
	#	% [5]	#	% [5]		
0	70.960,000	71.7	1,233,700	26.8	1.7	1.00
0–2	7,431,700	7.5	549,200	11.9	6.9	4.25
2–4	5,776,000	5.8	566,100	12.3	8.9	5.64
4–6	4,955,800	5.0	749,500	16.3	13.1	8.70
6–8	3,235,600	3.3	431,800	9.4	11.8	7.68
Over 8	6,669,300	6.7	1,072,200	23.3	13.9	9.25
Total	99,028,400		4,602,500		4.4	

[1] Numbers estimated by NIOSH using data from the 1988 NHIS–OHS conducted by NCHS (Exs. 26–1104, 26–1105, 26–1106).
[2] Estimated number of currently employed workers experiencing no episodes of back pain every day for 1 week or more during the 12 months prior to the survey.
[3] Estimated number of currently employed workers experiencing an episode of back pain every day for 1 week or more due to repeated activities at their current or most recent job during the 12 months prior to the survey.
[4] The odds ratio approximates the risk of an episode of back pain lasting 1 week or more due to repeated activities at work for workers engaged in strenuous physical activity such as listing, pushing, or pulling relative to the risk of an episode of back pain for workers with no such exposure.
[5] Percentage may not add to 100 due to rounding.

Table V–4.—Estimated Number of Currently Employed Workers Engaged in Repeated Bending, Twisting, or Reaching, by Duration and Back Pain Status [1]

HOURS ENGAGED	BACK PAIN				PERCENT	ODDS RATIO [4]
	NONE		AT LEAST 1 WEEK DUE TO REPEATED ACTIVITIES AT WORK [3]			
	#	% [5]	#	% [5]		
0	57,020,000	58.1	501,100	11.0	0.9	1.00
0–2	5,664,100	5.8	288,200	6.3	4.8	5.79
2–4	7,478,000	7.6	553,500	12.2	6.9	8.42
4–6	8,088,800	8.2	736,600	16.2	8.3	10.36
6–8	6,556,800	6.7	766,500	16.9	10.5	13.30

Table V–4.—Estimated Number of Currently Employed Workers Engaged in Repeated Bending, Twisting, or Reaching, by Duration and Back Pain Status [1]—Continued

HOURS ENGAGED	BACK PAIN				PERCENT	ODDS RATIO [4]
	NONE		AT LEAST 1 WEEK DUE TO REPEATED ACTIVITIES AT WORK [3]			
	#	% [5]	#	% [5]		
Over 8	13,340,000	13.6	1,697,100	37.4	11.3	14.08
Total	98,148,600		4,543,000		7.1	

[1] Numbers estimated by NIOSH using data from the 1988 NHIS–OHS conducted by NCHS (Exs. 26–1104, 26–1105, 26–1106).

[2] Estimated number of currently employed workers experiencing no episodes of back pain every day for 1 week or more during the 12 months prior to survey.

[3] Estimated number of currently employed workers experiencing an episode of back pain every day for 1 week or more due to repeated activities at their current or most recent job during the 12 months prior to the survey.

[4] The odds ratio approximates the risk of an episode of back pain lasting 1 week or more due to repeated activities at work for workers engaged in repeated bending, twisting, or reaching relative to the risk of an episode of back pain for workers with no such exposure.

[5] Percentage may not add to 100 due to rounding.

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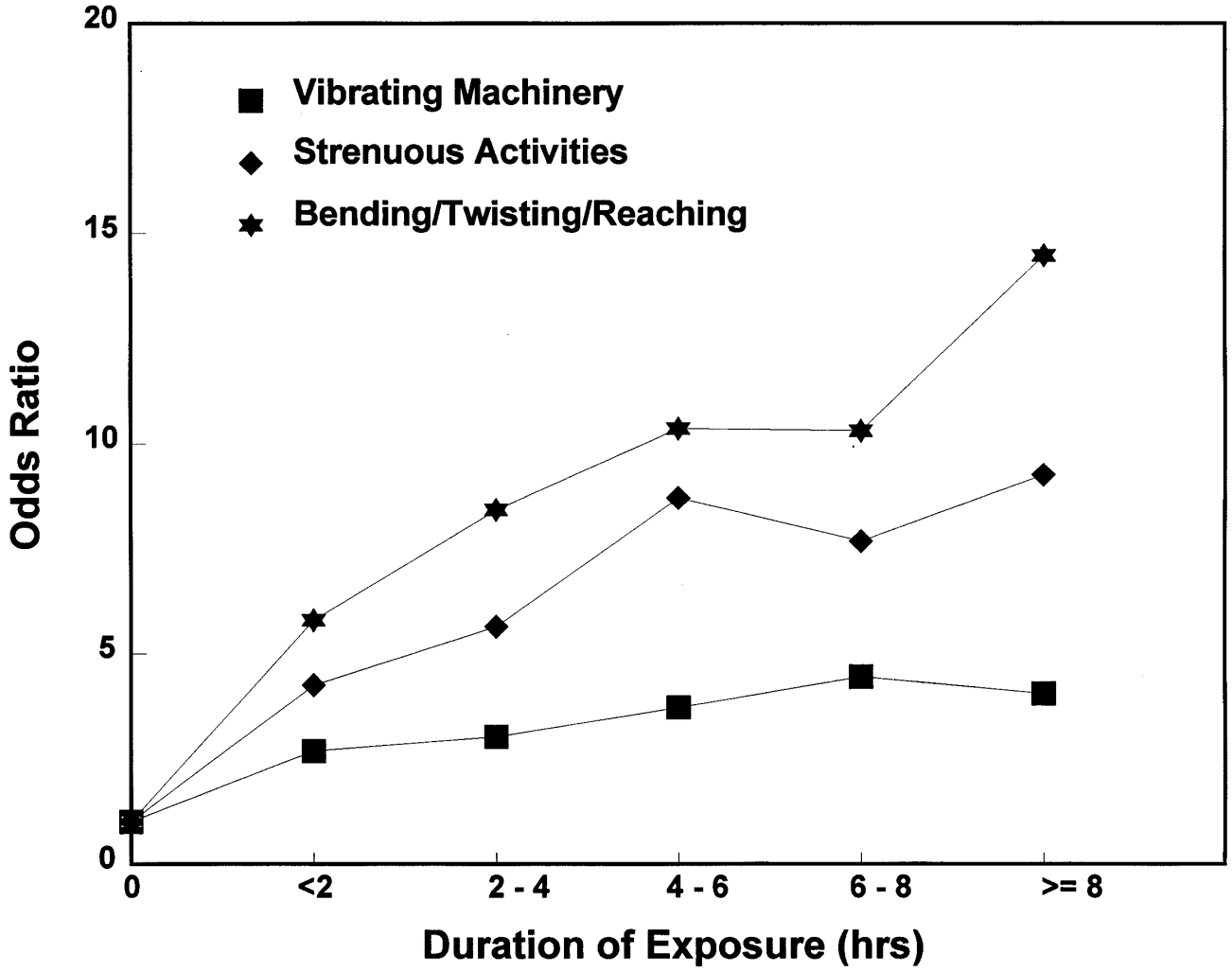
Figure V-1.
Relationship Between Duration of Exposure and Risk of Back Pain



Source: NIOSH analysis of data (Exs. 26-1104, 26-1105, 26-1106, 26-1107) from the National Health Interview Survey conducted by the National Center for Health Statistics.

Note: The odds ratio approximates the risk of an episode of back pain lasting 1 week or more due to repeated activities at work relative to the risk of an episode of back pain for workers with no such exposure. Work-related exposures include strenuous physical activity; repeated bending, twisting, or reaching; or the hand operation of vibrating machinery. Data excludes bac pain reported from acute injury or trauma.

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The ORs calculated from the data provided by NIOSH are very conservative. It is highly likely they underestimate the true ORs for the currently employed population. Only workers suffering episodes of back pain due to repeated activities at their current or most recent job are included. Workers who suffered episodes of back pain at a previous job are excluded. Workers who suffered episodes of back pain due to repeated activities on the job and due to an accident are also excluded. Finally, as observed by Bernard *et al.* (1993, Ex. 26–439), workers tend to overestimate the amount of time they spend daily at specific activities, particularly when such activities are hard and/or painful. Therefore, exposure is likely to be overestimated, meaning that risks at the lower exposure levels are likely to be underestimated. Despite the limitations of this analysis, the NCHS data clearly show a relationship between episodes of back pain lasting 1 week or longer and duration of exposure to workplace risk factors.

A similar analysis was conducted by Punnett *et al.* (1991, Ex. 26–39), using data from a case-control study of automobile assembly workers. To determine the relationship between back disorders and both postural stress and daily duration of exposure, the authors estimated the ORs from a logistic regression analysis. Duration of exposure was divided into two categories: 0 to 10% of cycle time and 10% or more of cycle time. Three types of postural stress were examined: any postural stress, mild flexion, and severe flexion. The results of this study, presented in Table V–5 and Figure V–2, show that for any postural stress and for mild flexion, the risk of back disorders was approximately 1.4 times greater for workers exposed for 10% or more of cycle time compared to workers exposed less than 10% of cycle time. For severe flexion, the risk of back disorders was approximately 2 times greater for workers exposed for 10% or more of cycle time than it was for workers exposed less than 10% of cycle time. The greatest increase in risk was seen among workers exposed to severe trunk flexion for more than 10% of cycle time (OR = 8.9 compared to unexposed workers). Thus, this study suggests that reductions in severity or duration of exposure to awkward trunk postures, even where exposure cannot be eliminated, may reduce risk of back disorders up to 2-fold.

Holmstrom, Lindell, and Moritz (1992, Ex. 26–36) estimated age-standardized prevalence rate ratios to examine the relationship between duration of exposure to different working postures and low-back and neck/shoulder pain in construction workers. Age standardization ia a statistical approach that controls for the effect of age on the health outcome being studied. This is usually done by selecting control subjects that match the ages of the individuals in the study cohort, or by using standardized illness rates for local or national populations. Controlling for age permits the investigator to compare the effect of age on the health outcome of interest with the effect of other variables, such as degree of exposure to a hazard. The age-standardized prevalence ratio is comparable to an age-adjusted odds ratio.

Table V–5.—Estimated Odds of Back Disorders in Workers With Varying Durations and Severities of Exposure [1]

TRUNK POSTURE	PERCENT OF CYCLE TIME	ODDS RATIO
Any posture	0–10%	3.8
	>10%	5.5
Mild Flexion	0 to 10%	4.2
	>10%	6.1
Severe Flexion	0 to 10%	4.4
	>10%	8.9

[1] Punnett *et al.,* 1991, Ex. 26–39.

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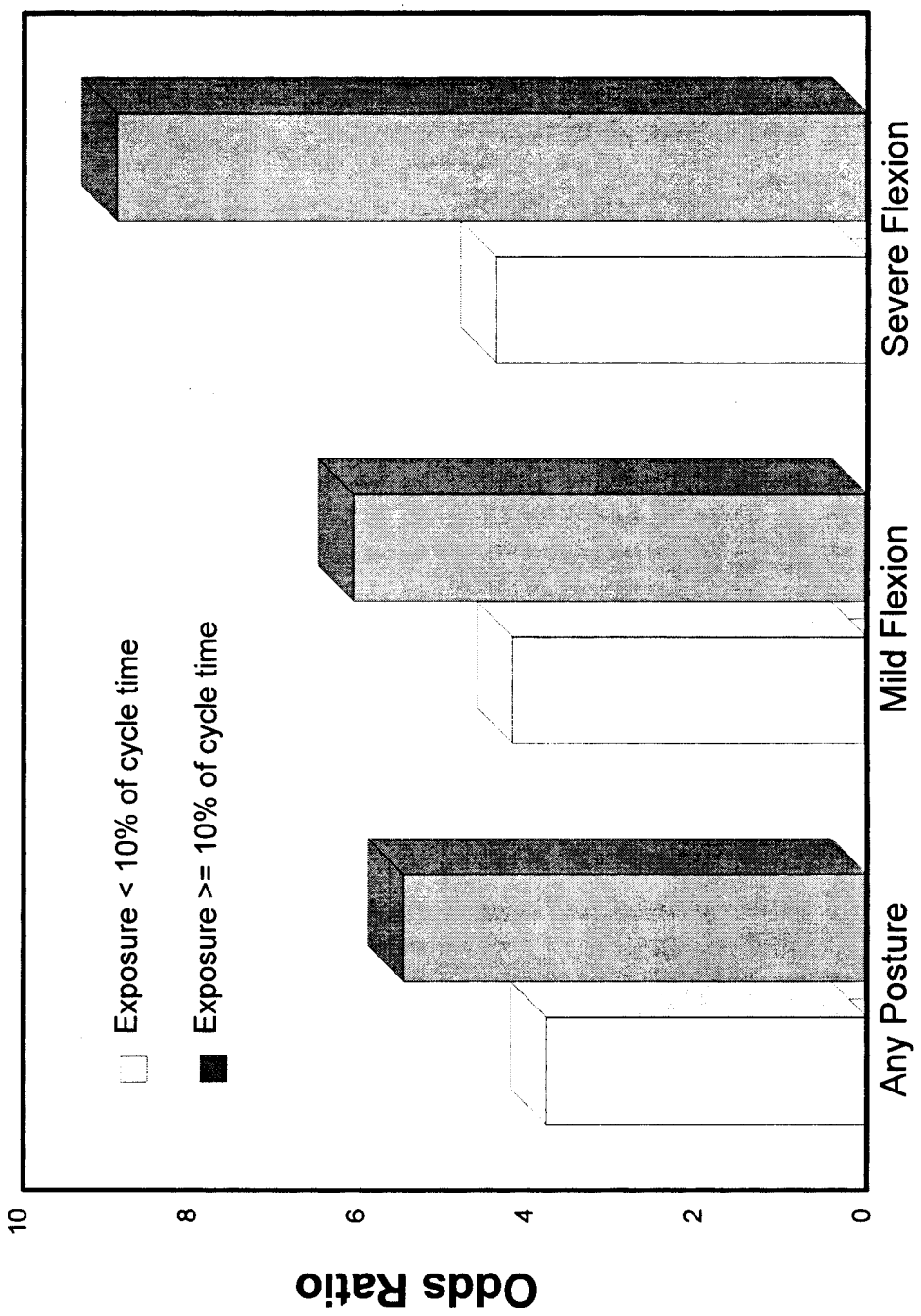
Figure V-2.
Relationship Between Duration and Severity of Exposure and Risk of Back Disorders



Source: Punnett et al., 1991 (Ex. 26-39)

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The results of the Holmstrom study are presented in Tables V–6 and V–7, and in Figure V–3. Three working postures were found to be associated with low-back pain: hands above shoulder level, stooping, and kneeling. In each case, the risk of severe back pain increases with exposure, with the largest increases in risk being associated with more than 4 hours per day of exposure to kneeling or stooping. Table V–6 shows that the greatest risk, associated with kneeling more than 4 hours per day, is 3.5 times greater among exposed workers than among workers with no exposure. These three working positions are also associated with considerable neck/shoulder pain. For this outcome, risk increases with duration of exposure as well. Table V–7 shows that for neck/shoulder pain, however, the greatest risk is associated with a posture of hands above shoulder level for more than 4 hours per day.

Table V–6.—Estimated Prevalence Rate Ratios of Severe Low-Back Pain in Construction Workers Engaged in a Variety of Postures, by Duration of Exposure [1]

POSTURE	HOURS OF EXPOSURE PER DAY	ODDS RATIOS	CONFIDENCE INTERVAL
Hands Above Shoulder Level	<1	1.09	0.8–1.5
	1–4	1.46	1.1–2.0
	>4	1.61	1.0–2.6
Stooping	<1	1.31	0.9–1.8
	1–4	1.88	1.4–2.6
	>4	2.61	1.7–3.8
Kneeling	<1	2.4	1.7–3.3
	1–4	2.6	1.9–3.5
	>4	3.5	2.4–4.9

[1] Holmstrom, Lindell, and Moritz, 1992, Ex. 26–36.

Table V–7.—Estimated Prevalence Rate Ratios of Neck/Shoulder Pain in Construction Workers Engaged in a Variety of Postures, by Duration of Exposure [1]

POSTURE	HOURS OF EXPOSURE PER DAY	ODDS RATIOS	CONFIDENCE INTERVAL
Hands Above Shoulder Level	<1	1.1	0.8–1.5
	1–4	1.5	1.2–1.9
	>4	2.0	1.4–2.7
Stooping	<1	1.0	0.8–1.3
	1–4	1.4	1.1–1.8
	>4	1.5	1.1–2.1
Kneeling	<1	1.4	1.1–1.8
	1–4	1.4	1.1–1.8
	>4	1.5	1.1–2.1

[1] Holmstrom, Lindell, and Moritz, 1992, Ex. 26–36.

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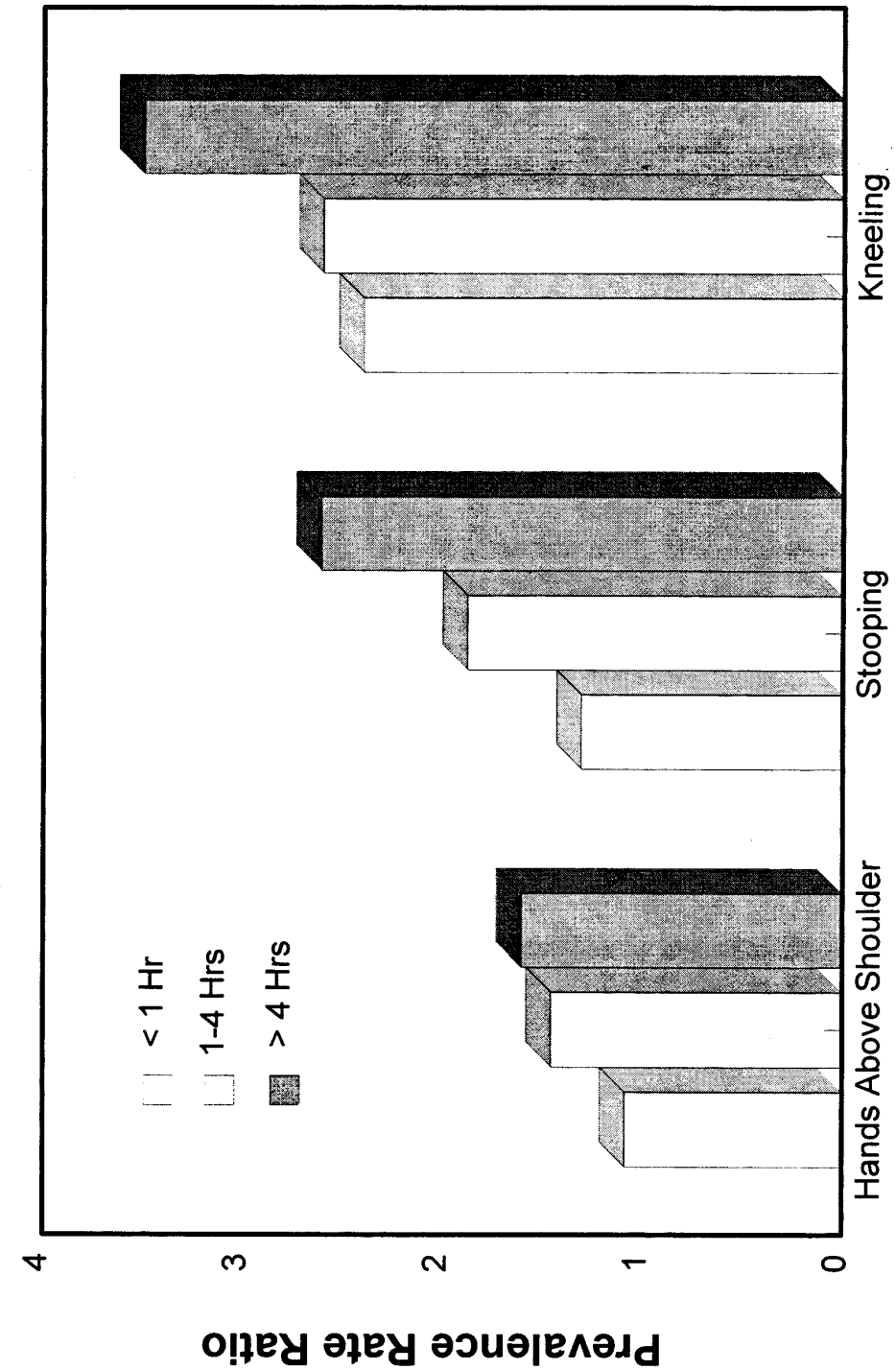
Figure V-3
Relationship Between Duration of Exposure, Type of Risk Factor, and Risk of Severe Lower Back Pain



Source: Holmstrom et al., 1992 (Ex. 26-36)

Note: Severe lower back pain defined as pain lasting at least 8-30 days over the past year and with "very severe" functional impairment.

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A prospective study by Liles *et al.* (1984, Ex. 26–33) demonstrated a clear relationship between intensity of exposure to manual handling risk factors and incidence of both total and lost-work-day back injuries. The study is unusual in that healthy workers were followed for over 1 year to determine the annual rate of back disorders. Exposure to manual handling risk factors was measured using a job severity index (JSI). A JSI is a measure of musculoskeletal strain based on weight handled, frequency of lifting, and a worker's physical capacity for lifting. A JSI of 1 or less means that the work task involved handling loads at or less than the worker's physical capacity for lifting. There was no apparent increase in either total or lost-work-day back injuries among workers whose jobs scored below a JSI of 1.5. Above this level, both total and lost-work-day injury rates increased dramatically, about 5-fold. The authors interpreted this finding as indicating that there is a threshold exposure level for back injuries due to manual handling and that back injuries can be expected to increase when workers handle loads exceeding their capacities by 50%. These data also suggest that back injury rates can be reduced by as much as 5-fold in manual handling tasks if they are designed to impart a physical load below 1.5 times the physical capacity of the worker, either by reducing duration of exposure or by reducing load weights or geometries. Figure V–4 graphically presents the relationship between the JSI and back injury rates.

Exposure-response relationships have also been demonstrated for upper-extremity MSDs. As with back disorders, studies have demonstrated that the risk of these illnesses increases dramatically with increasing daily duration of exposure to risk factors. For example, de Krom *et al.* (1990, Ex. 26–102) used ORs from a case-control study to assess the relationship between duration of exposure and MSDs. The authors estimated ORs from a logistic regression analysis that controlled for sex, age, and the interaction between age and sex to determine whether there was a relationship between CTS and the amount of time workers were engaged weekly in activities requiring a flexed wrist position, and between CTS and the amount of time workers were engaged weekly in activities requiring an extended wrist position. The results of this study, presented in Table V–8 and in Figure V–5, show that for both of these workplace risk factors—activities requiring a flexed wrist position and activities requiring an extended wrist position—the risk of CTS clearly increases as the number of hours spent each week in these activities increases.

Table V–8.—Estimated Odds of Carpal Tunnel Syndrome in Workers Engaged in Flexed Wrist and Extended Wrist Activities, by Duration of Exposure [1]

ACTIVITY	HOURS OF EXPOSURE PER WEEK	ODDS RATIOS	CONFIDENCE INTERVAL
Flexed Wrist	0	1.0	
	1–7	1.5	1.3–1.9
	8–19	3.0	1.8–4.9
	20–40	8.7	3.1–24.1
Extended Wrist	0	1.0	
	1–7	1.4	1.0–1.9
	8–19	2.3	1.0–5.2
	20–40	5.4	1.1–27.4

[1] de Krom *et al.,* 1990, Ex. 26–102.

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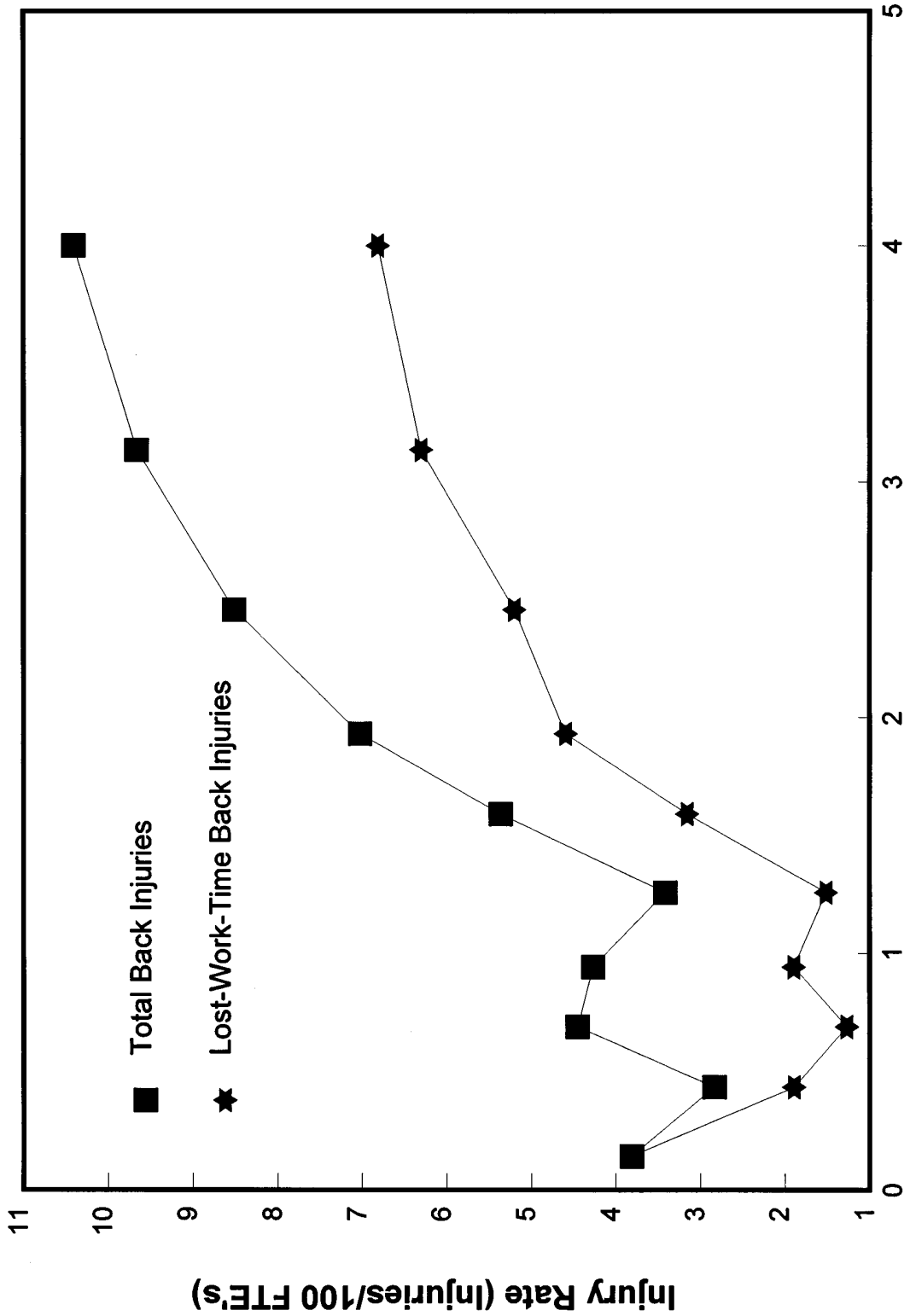
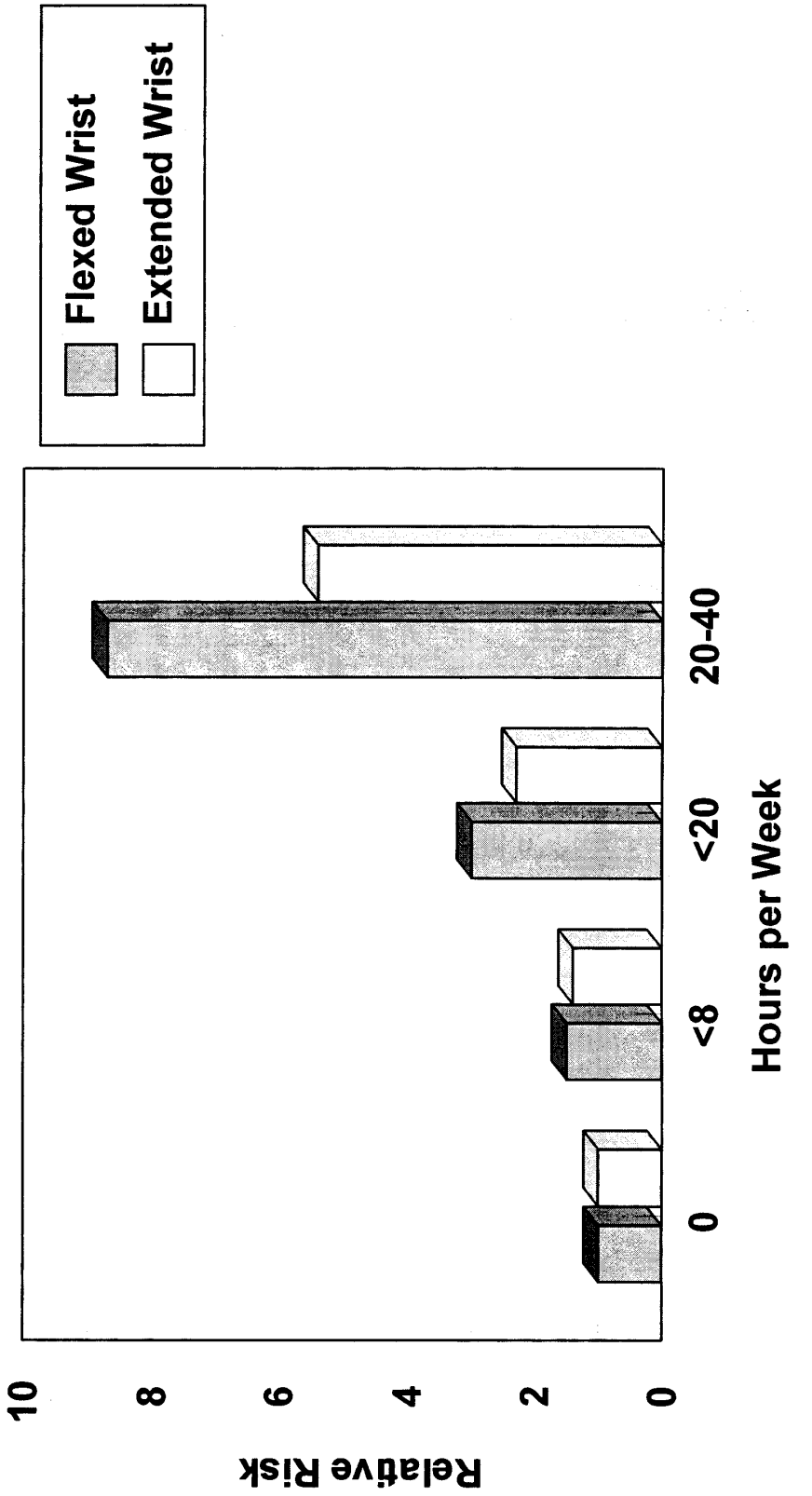
Figure V-4
Cumulative Injury Rate vs. Job Severity Index



Source: Liles et al., 1984 (Ex. 26-33)

Figure V-5
Relationship Between Duration of Exposure to Flexed or Extended Wrist and Relative Risk of Carpal Tunnel Syndrome



Source: deKrom et al., 1990 (Ex. 26-102)

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For workers engaged in activities requiring flexed wrists for as few as 8 to 19 hours per week (averaging approximately 1.5 to 4 hours per day), the odds of suffering CTS were three times greater than for workers engaged in activities that did not require flexed wrists. In contrast, the odds of suffering CTS in workers with average daily exposure to activities requiring flexed wrists in excess of 4 hours per day was 8.7 times greater than in workers with no exposure, or almost 3 times greater than for workers exposed less than 4 hours per day. Similarly, for workers engaged in activities requiring extended wrists for as few as 8 to 19 hours per week, the odds of suffering CTS were 2.3 times greater than for workers engaged in activities that did not require extended wrists. The odds of suffering CTS in workers with average daily exposure to activities requiring flexed wrists in excess of 4 hours per day was 5.4 times greater than in workers with no exposure. Thus, for workers engaged in tasks involving flexed or extended wrists for more than 4 hours daily, this study suggests that the risk of CTS can be reduced 2- to 3-fold by reducing daily exposure to less than 4 hours.

The duration of exposure to workplace risk factors is not the only factor associated with increased risk of work-related MSDs. Exposure to multiple workplace risk factors has also been found to be associated with increased risk. For example, in a study of workers at six industrial sites, Silverstein *et al.* (1986, Ex. 26–1404) studied the relationship between hand/wrist cumulative trauma disorders and exposure to activities requiring low force and low repetition, high force and low repetition, low force and high repetition, and high force and high repetition. Using logistic regression analysis to estimate ORs, these authors reported that the odds of suffering hand/wrist cumulative trauma disorders were 1.0 for workers engaged in low-force and low-repetition activity (*i.e.*, the control group), 3.3 for workers engaged in low-force and high-repetition activity, 5.2 for workers engaged in high-force and low-repetition activity, and 29.1 for workers engaged in high-force and high-repetition activity (see Figure V–6).

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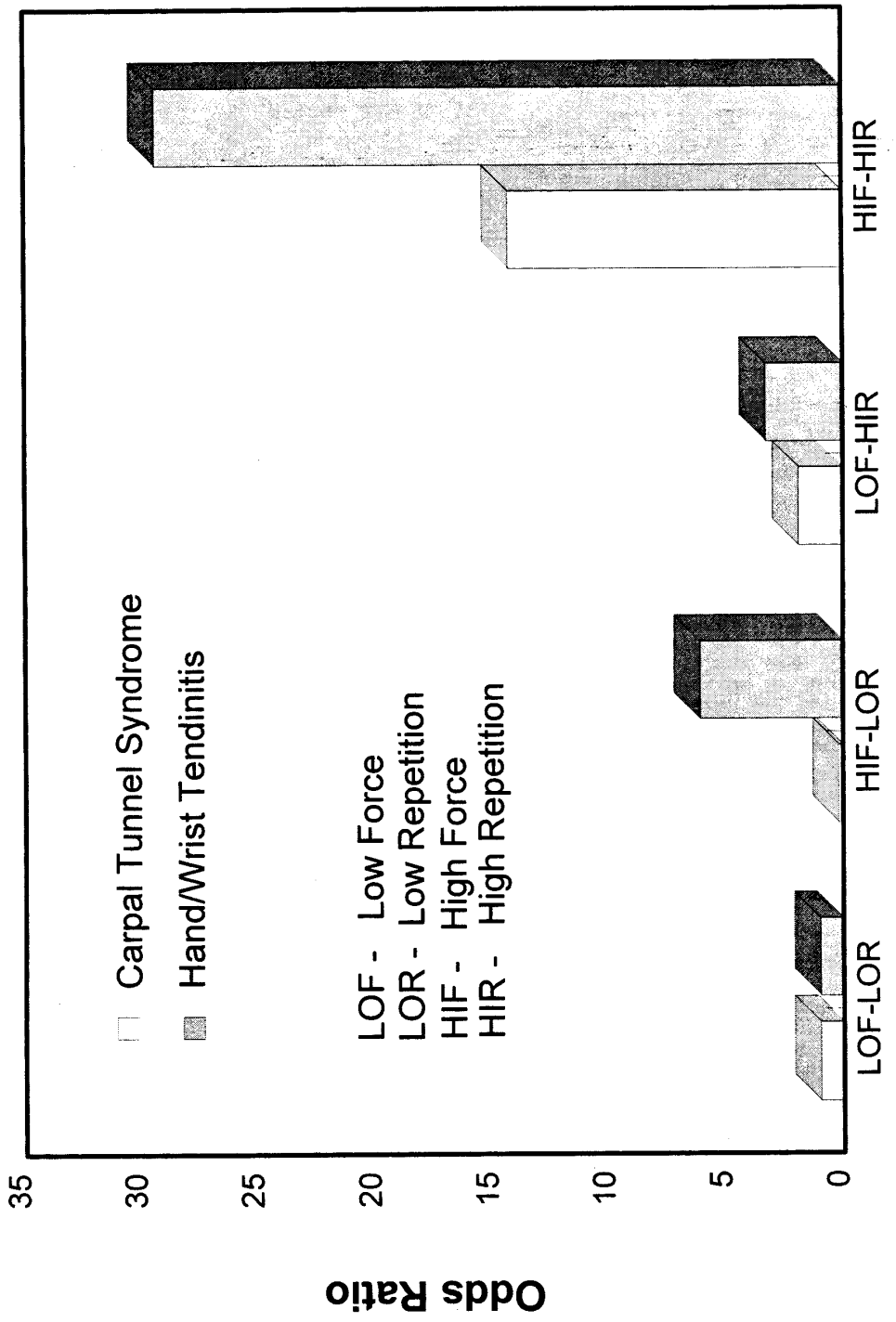
Figure V-6
Relationship Between Exposure to Risk Factors and Risk of Upper Body Musculoskeletal Disorders



Source: Armstrong et al., 1987 (Ex. 26-48); Silverstein et al., 1987 (Ex. 26-34)

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Similar findings for CTS were reported for workers in seven industrial sites (also shown in Figure V–6). Using logistic regression analysis to estimate ORs, these authors reported that the odds of suffering CTS were 1.0 for workers engaged in low-force and low-repetition activity (*i.e.*, the control group), 1.8 for workers engaged in high-force and low-repetition activity, 2.7 for workers engaged in low-force and high-repetition activity, and 15.5 for workers engaged in high-force and high repetition activity. Thus, the risk to workers exposed to two risk factors (high repetition and high force) was 7 to almost 10 times higher than the risk to workers exposed to only one risk factor. These data also suggest that risk increases more than linearly with increasing duration or intensity of exposure. Moore and Garg (1994, Ex. 26–1033) reported a similar finding among meat processing workers at risk for upper-extremity disorders. They found that the incidence of all upper-extremity disorders increased by the square of the amount of hand force applied in the job.

Loslever and Ranaivosoa (1993, Ex. 26–161) examined 17 jobs at high risk for CTS. For each job, they measured the amount of time the workers spent with flexed or extended wrists, the degree of flexion or extension, and the amount of force exerted. They found that the prevalence across jobs of CTS in both wrists increased in a dose-dependent manner as the combined exposure to force and flexion across jobs increased. In addition, the combination of force and flexion explained approximately 39% of the total variation in the prevalence of bilateral CTS across jobs.

Other supporting evidence for the existence of exposure-response relationships for upper-extremity disorders includes studies by Viikari-Juntura *et al.* (1994, Ex. 26–873) of neck disorders among machine operators, construction carpenters, and office workers, and a case-control study by English *et al.* (1995, Ex. 26–848) showing an exposure-response relationship between the rate of wrist flexion/extension and the ORs for disorders of the thumb.

Punnett (1998, Ex. 26–442) conducted a cross-sectional study in an automobile stamping plant and an engine assembly plant using an exposure-scoring protocol that reflected the intensity and duration of exposure to any of several workplace risk factors (*e.g.*, lifting/lowering, pushing/pulling, repetitive hand motion, awkward postures). The total exposure score had a possible range from 0 to 25 and was divided into quartiles, as indicated in Tables V–9 and V–10. The results are quite consistent, indicating that regardless of whether a case was defined by a physical examination or by self-reported symptoms, the prevalence of illness increased in a dose-dependent manner through exposure levels 13 to 18. Above that level, prevalence appears to hit a plateau. The author suggests that this plateau may be due to a ''healthy worker'' effect. By this she means that exposures at this level are so severe that workers move out of these jobs quickly, either to other jobs or to disability status. As a result of this relatively high turnover, healthy workers are frequently moved into these jobs. Thus the observed prevalence does not conform to a monotonic dose-response model.

Table V–9.—Prevalence Ratios for MSDs

[Based on Physical Exam]

EXPOSURE SCORE BASED ON CHECKLIST	SHOULDER/UPPER-ARM MSDs	HAND/WRIST MSDs	ALL UPPER-EXTREMITY MSDs
0–6	1.0	1.0	1.0
7–12	2.6	1.9	2.0
13–18	3.6	2.4	2.6
19–25	2.3	2.3	2.8

Table V–10.—Prevalence Ratios for MSDs

[Based on Symptom Reporting]

EXPOSURE SCORE BASED ON CHECKLIST	SHOULDER/UPPER-ARM MSDs	HAND/WRIST MSDs	ALL UPPER-EXTREMITY MSDs
0–6	1.0	1.0	1.0
7–12	2.5	2.0	1.8
13–18	3.8	2.5	2.4
19–25	3.5	2.5	2.3

Source: Punnett, 1998, Ex. 26–442.

Taken together, these studies provide compelling evidence of a causal relationship between exposure to workplace risk factors and an increased risk of developing MSDs. Furthermore, these studies demonstrate that the risk of work-related MSD can be substantially reduced by reducing the frequency or duration of exposure to any workplace risk factor, and by reducing the number of workplace risk factors to which workers are exposed.

6. Summary

The evidence summarized in this section is convincing and consistent. Studies from very different research traditions, and incorporating very different research

methodologies, strongly support the causal association of force, awkward postures, static postures, repetition, and vibration with work-related MSD outcomes. The evidence also strongly supports the effects of the four modifying factors on the impact of the exposures and the body's ability to repair the damage. The evidence is less strong in the case of external compression and dynamic factors, partly because of a relative shortage of studies in these areas. But the evidence that does exist is congruent.

In sum, although not all the epidemiological studies reviewed demonstrate significant associations, the overwhelming majority justify a conclusion that the risk factors noted in this section, with effects adjusted by the four modifying factors, cause or exacerbate work-related MSDs. The laboratory evidence in each case provides plausible and demonstrable biologic mechanisms through which these exposures can cause the anatomical and physiological changes characteristic of these disorders. The psychophysical evidence, relying on research that has linked subjective reports of fatigue, discomfort, and exertion to measurable disease rates in industry, further strengthens this conclusion.

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D. Pathogenesis and Pathophysiologic Evidence for Work-Related Musculoskeletal Disorders

1. Overview

An extensive body of scientific research and information has led to the conclusion that specific work factors, combinations of these factors, and modifying attributes or conditions contribute to the development and manifestation of work-related musculoskeletal disorders (MSDs). The term ''work-related'' refers to the performance of work tasks or working in a specific work environment that significantly contributes to the pathogenesis or manifestation of these multifactoral conditions (World Health Organization, 1985, Ex. 26–1040). The multifactoral nature of many of these MSDs, including the potential contribution of pre-existing or non-work factors to the pathogenesis of some work-related MSDs, is recognized. Other sections of this document present epidemiologic and biomechanical evidence that addresses the association of work factors and certain MSDs. This section describes the pathogenic and pathophysiologic mechanisms that establish the biological plausibility of the findings of the epidemiologic and biomechanical observations included in the earlier sections and in the Appendices (Ex. 27–1).

The pathogenesis of work-related MSDs can refer to either single, point-in-time injuries, associated with work tasks that result in activities in which tissue tolerance is acutely exceeded, or circumstances in which the performance of specific work tasks or combinations of tasks over a prolonged period of time results in small and repeated tissue damage to muscles, tendons, joints, or nerve structures (Association of Schools of Public Health/NIOSH, 1986, Ex. 26–1323; Putz-Anderson, Doyle, and Hales, 1992, Ex. 26–419; Rempel, Harrison, and Barnhart, 1992, Ex. 26–520). Work activities suggested as potential factors in the development or expression of work-related MSDs include high rates of task repetition; excessive force requirements; static postures; awkward work postures; vibration; cold temperatures; weight of loads lifted, pushed, or pulled; position of a load in relationship to the spinal axis; frequency and duration of materials handling task performance; hand coupling; dynamics of lifting (*e.g.*, muscle velocity and acceleration); lack of sufficient rest or recovery periods; overtime; piecework; and other issues (Armstrong, 1986, Ex. 26–928; Armstrong *et al.*, 1987, Ex. 26–48; Bergquist-Ullman and Larsson, 1977, Ex. 26–933; Chaffin and Park, 1973, Ex. 26–1115; Frymoyer *et al.*, 1980, Ex. 26–707; Johanning *et al.*, 1991, Ex. 26–1228; Klein *et al.*, 1984, Ex. 26–972; Marras *et al.*, 1993, Ex. 26–170; Rempel, Harrison, and Barnhart, 1992, Ex. 26–520; Silverstein, 1985, Ex. 26–1173; Silverstein, Fine, and Armstrong, 1986a, Ex. 26–1153, 1986b, Ex. 26–1404; Snook, Campanelli, and Hart, 1978, Ex. 26–35; Stock, 1991, Ex. 26–1010; Waters *et al.*, 1993, Ex. 26–521; Waters, 1994, Ex. 26–1403).

To accomplish motion and work, muscle, nerves, connective tissue, and skeleton are affected by a number of external and internal physical demands causing metabolic and compensatory tissue reactions. For example, the forceful, static, continuous, and/or repetitive demands made by manufacturing assembly work or manual materials handling can alter the function and integrity of specifically affected tissues. This can lead to the development and

clinical manifestation of MSDs such as tendinitis, epicondylitis, rotator cuff syndrome, or low-back pain. External demands can include direct pressure or tissue friction. As an illustration, prolonged or excessive force exerted over the base of the palm (by tools, handles, etc.) during assembly tasks can damage the median nerve in the palm, causing signs and symptoms of carpal tunnel syndrome (CTS). Internal responses can include inflammatory responses to tissue injury, neurochemical changes, and altered metabolism. For example, a lumbar disc herniation from repetitive lifting of heavy loads can compress a spinal nerve root, with subsequent nerve root edema, altered tissue metabolism, production of inflammatory mediators, and expressed signs and symptoms of lumbar radiculopathy.

The consequences of these external and internal demands associated with work activities can include a spectrum of symptoms or clinical findings, such as subtle or obvious inflammation, pain, swelling, restricted movement, and tissue damage diagnosed as muscle strain or tear, ligamentous or cartilage injury, tendinitis or tenosynovitis, bursitis, nerve entrapment, disc herniation, or degenerative joint or disc disease. This does not mean that a precise dose-response relationship between task factor exposure and disease exists for each of these work-related MSDs. Clear and consistent patterns exist, however, among the epidemiologic studies, biomechanical models, and pathogenetic and pathophysiologic explanations for many work-related MSDs (Gordon, Blair, and Fine, 1994, Ex. 26–1399; National Academy of Sciences, 1998, Ex. 26–37; Bernard and Fine, 1997, Ex. 26–1).

Factors specific to the individual can also affect the development and/or manifestation of pathology. These include, for example, preexisting injuries or illnesses (such as diabetes, degenerative joint disease, or rheumatoid joint disease); individual susceptibility to injury or tissue damage (related to anthropometric characteristics, physical conditioning, age, or genetics); and avocational activities or hobbies. These can interact in a complex fashion, such that work acts either as a causative, contributing, or accelerating factor in the development and/or manifestation of disease (Putz-Anderson, Doyle, and Hales, 1992, Ex. 26–419; Rempel, Harrison, and Barnhart, 1992, Ex. 26–520). However, although non-work risk factors can influence the development or expression of MSDs, their role is generally not as important as workplace risk factors because the duration and intensity of work are seldom matched in the non-work settings. Additional important considerations pertain to interactions between co-existing MSDs. For example, once an MSD is established, subsequent physical compensatory changes can further predispose an individual to the development of additional MSDs. When injury causes an altered posture, decreased range of motion, or weakness or ability to respond to tactile feedback to one joint or region, there is often increased risk of injury to another joint or region due to compensatory, increased loading. One example is the loss of tactile feedback from CTS, leading to greater hand force output that in turn contributes to the development of tendinitis or epicondylitis.

Section D.2 discusses the interaction between work demands and the responses of skeletal muscle, tendon, ligament, nerve, blood vessels, joint, and cartilage. It reviews the biological plausibility of an association between workplace factors and work-related MSDs of the spine and upper and lower extremities. It also considers the contributions of age, genetics, gender, cigarette smoking, and avocational activities to the pathogenesis and pathophysiology of work-related MSDs.

Section D.3 focuses on vibration. A separate section on vibration is included here because real specificity exists for this risk factor. Vibration can be reliably linked with specific outcomes: damage to vessels and small, unmyelinated nerve fibers in the fingers. In contrast, most of the other tissue disorders discussed in Section D result from a combination of exposures.

2. Pathogenesis and Pathophysiology of Work-Related Tissue Injury

a. Skeletal Muscle. There are several explanations for the development of work-related skeletal muscle disorders. Acute muscle tears, an extreme example of work-related skeletal muscle disorders, may develop when task demands exceed muscle tissue tolerance. While this may occur during any type of muscle contraction, it is much more common during eccentric contraction (*i.e.* during muscle lengthening to control, rather than initiate, an action), perhaps due to the nature of muscle recruitment of fibers with less oxidative capacity (Friden and Lieber, 1994, Ex. 26–546). Yet even low-force, static, or prolonged muscle activities commonly noted in a variety of manufacturing and office settings have the potential to cause or contribute to the development of work-related skeletal muscle disorders (Hagg, 1991, Ex. 26–427; Henneman and Olson, 1965, Ex. 26–139; Herberts *et al.*, 1984, Ex. 26–51; Jarvholm *et al.*, 1989, Ex. 26–967; Murthy *et al.*, 1997, Ex. 26–307; Sjogaard, 1988, Ex. 26–206; Sjogaard and Sjogaard, 1998, Ex. 26–1322). Muscle recruitment patterns with low-extension, repetitive, or static activities may selectively injure low-threshold and more easily recruited muscle fibers, which have been referred to as ''Cinderella fibers'' because of their constant activity (Henneman and Olson, 1965, Ex. 26–134; Lieber and Friden, 1994, Ex. 26–559). Alternatively, hypoxia and metabolic abnormalities (fatigue), inflammatory responses, inadequate rest pauses, and repair mechanisms appear to explain some of these skeletal muscle disorders associated with certain jobs or tasks (Armstrong *et al.*, 1993, Ex. 26–1110; Bigland-Ritchie, 1983, Ex. 26–76; Faulkner and Brooks, 1995, Ex. 26–1440; Herberts *et al.*, 1984, Ex. 26–51; Sjogaard, 1988, Ex. 26–206; Sjogaard and Sogaard, 1998, Ex. 26–1322). Electromyography (EMG) has helped researchers to better understand skeletal muscle responses to work tasks, estimate muscle loading with activity and intramuscular pressure generation, and comprehend the development of muscle fatigue (Chaffin, 1973, Ex. 26–876; Chaffin and Andersson, 1991, Ex. 26–420; Dolan *et al.*, 1999, Ex. 26–819; Lieber and Friden, 1994, Ex. 26–559; Nieminen *et al.*, 1993, Ex. 26–1382; NIOSH, 1992, Ex. 26–1325). In addition, at least one study has demonstrated a significant impact of ergonomic interventions on diminishing both EMG-observed trapezius loading and sick time due to skeletal muscle morbidity (Aarås, 1994a, 1994b, 1987, Exs. 26–892, 26–62, 26–1034).

Skeletal muscle is a highly evolved tissue with specialized contractile properties and an exceptional capacity to adapt and change. The bodybuilder's ability to rapidly build muscle bulk and the weakness and atrophy that come with prolonged bed rest or disuse are two examples of this ''plasticity.'' Individual muscle fibers have a unique capacity to convert chemical energy into a specific level of time-limited mechanical work (capacity and endurance). There are hundreds of skeletal muscles in the human body, each responsible for specific motions of bone and joints, that permit work performance. In the setting of normal physiologic responses, the central nervous system (CNS)

releases nerve impulses which activate motor units, causing muscle contraction, tendon tension, and movement of bones and joints. Each skeletal muscle is attached to a site of origin, transitions through a myotendinous junction, and attaches to bone as tendon, sometimes crossing joints along the way.

The components of each skeletal muscle include muscle fibers, connective tissue, and nerve endings. Muscle fibers, in turn, are composed of contracting elements called myofibrils. These myofibrils contain thin (actin, troponin, and tropomyosin proteins) and thick (myosin protein) filaments that slide over each other, resulting in muscle contraction. The myofilaments are arranged in compartments (sarcomeres) separated from each other by thin zones of dense material (Z-lines). Upon stimulation from a motor nerve impulse, altered muscle membrane permeability (depolarization) releases calcium ions, which subsequently create cross-bridging between muscle filaments and resultant contraction. Skeletal muscle is covered by a connective tissue called the epimysium, which is contiguous with the perimysium, a septum that separates the muscle into muscle fiber bundles. These muscle fiber bundles further subdivide into individual muscle fibers surrounded by an endomysium. The connective tissue permits the passage of blood vessels and nerves through the skeletal muscle to the muscle fibers, and also contributes to the mechanical characteristics of the muscle, especially with respect to resistance to stretching or deformation.

Peripheral nerves traverse the connective tissue to carry (motor) impulses from the CNS to the muscle, attaching at the neuromuscular junction. The functional unit of a muscle is called the motor unit, and is composed of motor neurons and the muscle fibers they control. Small motor units, with a nerve fiber controlling a few muscle fibers, are located in areas such as the hand where fine motor tasks are performed. These smaller units allow contraction at lower forces. Larger units are located in the leg, where a single nerve fiber can activate hundreds or thousands of muscle fibers to permit gross motor tasks. When a nerve impulse activates a motor unit, all of the fibers in that unit contract simultaneously. The response of the entire muscle depends on several factors. After a nerve impulse, a certain number of motor units will contract in response. As the impulse increases, more units are recruited and greater force results. When stimulation occurs prior to relaxation, a larger contraction (or summation) will evolve. The size, temporal sequencing, and frequency of the stimulus will determine if a muscle reaches maximal contraction, with responses maintained until stimulation ceases or fatigue occurs. Sensory feedback control occurs via muscle spindles that sense the length and speed of contraction or stretch of the muscle fibers.

Muscle power also depends on the composition of the fibers and muscle length. Type I (slow) fibers are smaller, have a large capacity for aerobic work, take a longer time to reach peak tension, and permit sustained, low-level muscle activity. Type II (fast) fibers quickly reach peak tension and help with short-duration, intensive activity. Type II fibers, however, fatigue quickly. With disuse, type II fibers are the first to atrophy (Chaffin and Andersson, 1991, Ex. 26–420). Skeletal muscles at their relaxed length generate the greatest amount of tension. At resting length, there is optimal overlap between the thick and thin filaments to permit maximal shortening. As the muscle contracts, there is greater overlap and less potential to contract further. When muscles are stretched, there is less overlap, and therefore, less tension can be generated (Chaffin and Andersson, 1991, Ex. 26–420). As discussed above, the amount and characteristics of the passive connective tissue in the specific muscle also determine the tension developed when muscles are stretched.

Individual muscle fibers have a unique capacity to convert chemical energy into a specific level of time-limited mechanical work (capacity and endurance). This chemical energy is transported in the form of activated phosphorylated molecules, primarily adenosine triphosphate (ATP). Energy release to accomplish muscle contraction is provided by the splitting off of a phosphate group from adenosine triphosphate (ATP), which converts the ATP to adenosine diphosphate (ADP). Phosphocreatine enables ADP to be converted back to ATP, thereby re-supplying the muscle fiber with energy and permitting the contraction to continue for brief periods. With persistent contraction, ATP resynthesis occurs under aerobic (with oxygen) or anaerobic (without oxygen) conditions. During low to moderate exertion, aerobic conditions predominate. The exhaustion of these energy stores can lead to fatigue, and in extreme cases, injury to the muscle tissue itself (Armstrong, Warren, and Lowe, 1994, Ex. 26–525; Chaffin and Andersson, 1991, Ex. 26–420; Lieber and Friden, 1994, Ex. 26–559). Heat is also generated and expended as a result of this metabolic activity.

Researchers have described several types of muscle contraction. In isometric (static) contraction, the external length of the muscle remains fixed, despite sliding of myofibrils. High muscle tension is generated because there is no expenditure of energy to shorten the muscle. During isotonic contraction, muscle length changes while the tension remains constant. Energy is expended to permit this change in muscle length to occur. Concentric contraction involves muscle shortening. An example of this is when the biceps muscle contracts and shortens during elbow flexion. Eccentric contraction describes contraction during muscle lengthening, as when muscle activity is required to control an action rather than to initiate it. Velocity of contraction affects the tension a muscle generates, with less force generated as the velocity of shortening increases. This relates to the length of the muscle, discussed above, and friction. Endurance depends on the composition of fibers and the percentage of maximal muscle force (Chaffin and Andersson, 1991, Ex. 26–420; Lieber and Friden, 1994, Ex. 26–559). At efforts under 15% of maximal force, endurance can reach 45 minutes (Lieber, 1992, Ex. 26–433). As muscle approaches 35% of maximal force, endurance time decreases to approximately two minutes, and as exertion approaches 100%, endurance time approaches zero (Chaffin and Andersson, 1991, Ex. 26–420, p. 49). Gradual exercise programs, however, have the capacity to improve muscle strength and endurance.

Muscle proteins allow muscle fibers to stretch and to elastically recoil to their resting length. If a muscle is stretched excessively, these mechanoelastic properties of muscle fiber are exceeded and observable physical damage is incurred. There is an important distinction between injuries that are the result of muscle activities that exceed these mechanoelastic capacities of muscle, and injuries that have their origins in activities that are below maximum muscle capacity. The latter may involve sequential or stereotyped patterns of work, whose execution becomes compromised by pain or fatigue. In fact, the bulk of modern work involves activities that neither challenge nor exceed the mechanical limits of muscle fibers.

The types of injury acquired during more routine function involve potentially complex metabolic and neurologic processes. Changes in muscle morphology and fiber type

(gene expression), in muscle fatigue and failure (metabolic function), and in loss of centrally mediated coordinated movement (dystonia) are all examples of the biochemical and neurologic origins of some types of muscle injury. These mechanisms, rather than gross patho-anatomic injury and repair, are a major focus of current research on work-related muscle injury.

Muscle tissue has a high intrinsic repair capacity and can effectively adapt to diverse biomechanical loads. Understanding the divergent paths of successful learning and adaptation or injury and degeneration requires an understanding of physiology (Pette, 1980, Ex. 26–1304).

There are three events associated with muscle injury. While injury related to mechanical contraction is usually caused by stretch (eccentric contraction), injury may also occur during muscle shortening (concentric contraction), or while maintaining the muscle at a constant level of stretch and tension (isometric contraction). The basic mechanism is a mismatch between external load and internal contractile capacity. This results in mechanical disruption between the sarcomeres along the Z-lines. The outcome is inflammation, the sensation of muscle soreness, and triggering of repair processes.

A second injury mechanism is fatigue, in which there is an activity-related perception of raised effort or an inability to sustain force. Muscle fatigue occurs when physical tasks require high-power, short-duration repetitive contractions, or when there are low-power, sustained or repetitive contractions (Faulkner and Brooks, 1995, Ex. 26–1410). Fatigue has consequences for task performance and includes both rapidly reversible and non-reversible manifestations.

As a muscle becomes fatigued, it produces a distinct electrical signal that can be picked up by electromyography (EMG). The EMG signal is measured by placing electrical transducers on the skin surface over the muscle, or by inserting a needle or small wire directly into the muscle. EMG measurements are most often taken where muscles are well-defined and accessible. EMG has other uses. EMG has been an important tool in measuring effort and fatigue in the large muscles of the neck and shoulders. Recorded EMG voltage reflects the sum of several motor unit potentials. The primary usefulness of surface EMG in work settings is to estimate muscle tension associated with task performance from measured myoelectric activity. Since many factors affect the relationship between muscle force and the amplitude of myoelectric activity, several methods are used to improve the correlation (Chaffin and Andersson, 1991, Ex. 26–420; Dolan *et al.*, 1999, Ex. 26–819; NIOSH, 1992, Ex. 26–1325). Individual-and activity-specific calibration can be performed by measuring myoelectric activity and external moments while a subject performs graded activity. Normalization can be employed by measuring one isometric maximum voluntary contraction (MVC) and reporting the activity as a percentage of MVC. This appears to correlate reasonably with load moments calculated from other models (Nieminen, 1993, Ex. 26–1382). Measurements of myoelectric activity can then be used to estimate load moments or forces during the performance of more complex tasks in a variety of work settings. Fatigue can also be assessed: muscle activity is observed to show an increased amplitude and decreased frequency in the myoelectric signal with fatigue (Chaffin and Andersson, 1991, Ex. 26–420; Chaffin, 1973, Ex. 26–876; Lieber and Friden, 1994, Ex. 26–559). This is consistent with laboratory observations of the response in fatigued muscle fiber (Bigland-Ritchie *et al.*, 1983); the authors hypothesize that this may be a physiologic adaptation'slower muscles are able to generate higher forces.

Dolan *et al.* (1999, Ex. 26–819) recently validated the usefulness of this technique in evaluating dynamic lumbar spine loading. The authors studied eight male subjects who performed lifting tasks from floor height (boxes weighing 6.7 and 15.7 kg). L5–S1 joint moments were assessed using force plates and by measuring the EMG activity of the erector spinae muscles. The two assessment methods yielded similar peak extensor moments, equivalent to spinal compressive forces of 2.9 to 4.8 kN. The researchers did note, however, that there were small deviations during lifts requiring a vigorous upward thrust from the legs, and that additional force-plate data would mildly improve correlation in these settings.

A third injury mechanism (after mechanical contraction-related injury and muscle fatigue) is the release of neuro-humoral substances and changes in electrolyte balance. Neuro-humoral substances are chemicals that affect cell membranes and cell function and excite afferent nerves. Muscle pain, inflammation, and ischemia, or sustained static contraction, lead to release of potassium chloride, lactate, arachidonic acid, bradykinins, serotinin, and histamine. In addition to producing pain, these agents can excite chemosensitive afferents—gamma muscle spindles—that respond to stretch. It is hypothesized that increased spindle excitation can cause the stiffness and pain of ''myalgia'' (Johannson and Sojka, 1991, Ex. 26–968). There is substantial evidence that these mechanisms of tissue injury can produce a distinct MSD pattern, particularly when the work stressors are not sufficiently intense to produce outright mechanical injury. At even 10% of MVC, muscle oxidation declines significantly (Murthy *et al.*, 1997, Ex. 26–307). Proprioceptive accuracy and efficiency are also significantly limited under conditions of fatigue. The loss of accuracy and fine control in hand-intensive tasks, such as manual tool use, requires greater muscle recruitment and correction, further increasing demands on muscle.

Several mechanical and physiologic muscle responses are involved in the generation of muscle forces and motion of skeletal structures that relate to the development of pathology. Coordination of muscle activity to manipulate bones and joints involves initiation by agonist muscles, with regulatory contributions from synergistically and antagonistically acting muscles. The forces generated by these muscles around a joint produce load moments on the joint. This can cause compression or rotation at the joint with secondary effects on the joint cartilage or bone.

An acute muscle tear is a point-in-time injury that results when the force demands exceed the muscle tissue mechanical tolerance. This can occur during rapid intentional movement or during a loss of balance, such as in a fall. Often there is rapid stretching of muscle in addition to contraction (Lieber and Friden, 1993, Ex. 26–160), and injuries are generally worse when muscle is in its stretched position (Macpherson, Shork, and Faulkner, 1996, Ex. 26–165). Healing requires 1 to 4 weeks (Ashton-Miller, 1999, Ex. 26–414; Brooks and Faulkner, 1990, Ex. 26–85), and there is potential for decreased strength after healing is achieved.

After injury, satellite cells proliferate to repair the muscle damage. As people age, fewer satellite cells are observed in muscle tissue; this may explain the delayed recovery in injured older workers (Carlson, 1994, Ex. 26–530). However, muscle rupture may also occur when mechanical disruption of sarcomeres produces an inflammatory response (free radicals, cytosolic enzymes, phagocytosis) with an increased

susceptibility to delayed muscle tear (Faulkner and Brooks, 1995, Ex. 26–1410).

Reduced blood flow and increased transmural muscle pressure appear to be important predisposing factors to injury (Armstrong *et al.*, 1993, Ex. 26–1110; Kilbom, 1994, Ex. 26–1352; Sjogaard and Sogaard, 1998, Ex. 26–1322). The reduced blood flow that is characteristic of static contraction and increased transmural pressure is reversible. However, there is additional evidence that the pattern of reduced flow, injury and diminished repair, and chronic fiber damage all contribute to muscle pain (Lindman *et al.*, 1991, Ex. 26–976). Sufficient blood flow to skeletal muscle is essential for contraction, since force development depends on the conversion of chemical to mechanical energy. EMG studies show increased EMG activity in repetitive and stereotyped work in the setting of myalgia. All of this points to the particular problems of continued use of muscle that has already sustained injury, since the normal processes of adequate blood supply and oxygenation, ability to sustain contraction, and the capacity for repair are all compromised. Prolonged skeletal muscle contraction can produce other complications related to elevated intramuscular pressure. Secondary ischemia and disruption of the transportation of nutrients and oxygen can produce intramuscular edema (Sjogaard, 1988, Ex. 26–206). This is compounded when recovery time between contractions is insufficient. Eventually, muscle membrane damage, abnormal calcium homeostasis, free radicals, other inflammatory mediators, and degenerative changes can occur (Sjogaard and Sjogaard, 1998, Ex. 26–1322).

It is also important to recognize that sustained injury appears to involve the excitation of specific neural pathways, rather than occurring as the result of simple repetitive tonic activities. The implications are that simple overuse is remediable and apparent functional loss is often a protective mechanism against depleting muscle cells' energy stores. However, more complex muscle injury involves changes in nerve-muscle interaction and inflammatory changes, and continued use and insult can cause more chronic aggravation.

Several studies appear to support belief in these pathogenic mechanisms. Veiersted *et al.* (1993, Ex. 26–1154) performed EMG studies on subjects performing machine-paced packing work. Individuals with symptoms of trapezius pain had fewer rest pauses and a shorter total duration of rest pauses, suggesting higher levels of muscle fiber activity. Aarås (1987, Ex. 26–1034) demonstrated that reduction of trapezius muscle activity to less than 2% of MVC in assembly workers reduced sick time. Interesting pathophysiologic findings were noted by Larsson *et al.* (1990, Ex. 26–1141) when they evaluated trapezius muscle biopsies and blood flow in assembly workers with localized chronic myalgia related to static loading during assembly work. In symptomatic workers, reduced muscle blood flow and pathologic changes (ragged red fibers indicating disturbed mitochondrial function were confined to the type I fibers) were recorded. Myalgia was correlated with reduced local blood flow and the presence of mitochondrial changes.

Other authors have noted elevated serum levels of muscle enzymes, particularly creatine kinase, in delayed onset muscular soreness following unaccustomed muscle exertion (Armstrong, 1990, Ex. 26–703; Newham *et al.*, 1983a, Ex. 26–395; Schwane *et al.*, 1983, Ex. 26–716). This is followed by degenerative changes in sarcomeres followed by regeneration and repair within about 2 weeks (Newham *et al.*, 1983b, Ex. 26–741; Ogilvie *et al.*, 1988. Ex. 26–189).

It must also be appreciated that work does not have to be repetitive or forceful to cause MSDs. Static postures involve repeated and prolonged low force contraction of low-threshold motor units. Although the total workload is low, the individual muscles and muscle fibers may approach their maximal capacity, which can lead to injury (Hagg, 1991, Ex. 26–427). For example, intramuscular pressures associated with static muscle contraction have the potential to cause muscle tissue injury. The magnitude of intramuscular pressure varies significantly depending on individual muscle characteristics (there are greater pressures in contracting bulky muscles as opposed to thin ones) and location (constricting fascial compartments and adjacent bony structures may increase pressures reached during contraction) (Sjogaard and Sogaard, 1998, Ex. 26–1322). Muscle activity and position also determine intramuscular pressures. Herberts *et al.* (1984, Ex. 26–51) demonstrated that increased hand loads and larger degrees of arm elevation will increase EMG activity and intramuscular pressures in shoulder girdle muscles (deltoid, infra- and supraspinatus, trapezius). This may be noted during static work tasks adopted to stabilize hand tools near shoulder heights during assembly or construction. While very forceful muscle contractions may produce intramuscular pressures that exceed systemic blood pressure, supravenous intramuscular pressures exceeding 40 to 60 mm Hg have even been observed in the supraspinatus muscle during static contractions of less than 10% of MVC (Jarvholm *et al.*, 1989, Ex. 26–967; Sjogaard *et al.*, 1996, Ex. 26–213). Therefore, muscle pressures during low-force static work may approach the range of diastolic pressures. Of importance, diastolic pressures are more significant than mean blood pressures for maintaining blood flow in low-flow situations (Sjogaard *et al.*, 1986, Ex. 26–207), resulting in the potential for damage to muscle tissues. The mechanism of muscle injury associated with elevated intramuscular pressures relates to secondary abnormalities of microcirculatory regulation caused by these pressure increases. As a result, several changes are noted. Diminished oxygen supply to muscle tissue will reduce its capacity to convert chemical to mechanical energy. Persistent contraction may increase tissue edema, potentially increasing tissue pressures and further impairing microcirculation.

In other circumstances, the recruitment of only a limited number of fibers can result in high fiber stress distributed across the few fibers involved in the contraction, although total muscle forces may be low. Because highly repetitive tasks can only be sustained for prolonged periods when low force is involved, type I fibers are more likely to be involved in repetitive injury.

Increasing attention has been paid to metabolic and neuroregulatory factors to better understand the relationship between acute muscle fatigue and the development of chronic muscle disorders, as well as to characterize the pattern of pain symptoms that affect the neck, shoulders, forearms, wrists, and fingers in manually intensive tasks that occur well below the MVC. Higher subjective levels of fatigue as well as electrophysiological evidence of fatigue are more common in large muscle groups, such as the neck and shoulder muscles, when activities are static and repetitive rather than dynamic (Sjogaard, 1988, Ex. 26–830). During low levels of exertion, skeletal muscle recruitment primarily activates the slower and less fatigable type I muscle fibers because of their lower thresholds (Henneman and Olson, 1965, Ex. 26–139) Lieber and Friden (1994, Ex. 26–559) have demonstrated an activation sequence by which these smaller, more fatigue-resistant muscle units are first

recruited, followed by stronger, more easily fatigued fibers. These smaller fibers are the ''Cinderella fibers,'' so named because they are always working in lower-threshold activity, which can be insufficient to recruit stronger fibers (Henneman and Olson, 1965, Ex. 26–139).

The concerns with sustained low-level activity are multifold. Limited muscle fiber recruitment can result in higher individual fiber stresses distributed across the few fibers involved in the contraction, although total muscle forces may be low. Because highly repetitive tasks can only be sustained for prolonged periods of time when low force is involved, type I fibers are more likely to be involved in repetitive injuries. The prolonged recruitment of limited numbers of motor units, even during situations with low stress on these muscle fibers, can deplete available energy, producing eventual fatigue and injury (Lieber and Friden, 1994, Ex. 26–559). At low contraction levels, membrane resting potential is maintained in all fibers, including activated fibers (Sjogaard *et al.*, 1996, Ex. 26–213). Potassium-flux—induced fatigue is an important homeostatic mechanism for protecting essential ATP stores, but this essential mechanism is bypassed at lower activity levels. A fatigued muscle that will not contract prevents direct tissue damage. Otherwise, the infusion of cytosolic calcium continues. Although calcium is essential for contraction, its build-up is directly damaging to membrane lipids and mitochondria. There is mounting evidence that types of lower-output activity that bypass homeostatic protection can dispose active muscle to silent but significant injury. Skeletal muscle recruitment may also explain the observation that eccentric muscle contraction more commonly causes muscle injury than does concentric contraction (Friden and Lieber, 1994, Ex. 26–559), since this type of contraction primarily involves the fastest fibers with the lowest oxidative capacity.

Finally, age effects on skeletal muscle generally result in greater susceptibility to injury with repeated loading. With aging, muscle contractility is diminished (Thelen *et al.*, 1996a, Ex. 26–219), muscle mass and maximum isometric force declines (Faulkner and Brooks, 1995, Ex. 26–1410), and the rate of developing force and power is lower (Thelen *et al.*, 1996b, Ex. 26–220). In older individuals, physical conditioning has more impact on power than it does on force. Age-related changes appear to be an intrinsic function of muscle fibers themselves, rather than a change in muscle recruitment patterns. Injuries from eccentric contractions in older animals heal more slowly and show a greater force deficit (injury effect) than in younger animals.

In summary, a significant body of evidence supports the conclusion that conditions often present at work can be pathogenetic and pathophysiologic links with many muscular disorders. There is strong physiologic evidence that sub-maximal muscle contraction, which is the prevailing pattern in the American manufacturing and office workplace, can produce patterns of chronic muscle injury. Potential etiologies include abnormalities in motor unit recruitment, tissue loading in susceptible positions, altered muscle metabolism and blood flow, energy depletion and fatigue, inflammation, and altered tissue repair. This is especially true when work evolves away from tasks that approach the limit of contractile forces, and specific pathways of injury, rather than force itself, become the critical elements in understanding disease. Applying ergonomic principles to muscle physiology is intended to preserve mechanical output while preventing tissue injury.

b. Tendons and Ligaments. Work-related tendon disorders develop for several reasons. Tendon has viscoelastic properties that may be exceeded when workers perform excessively forceful work activities, carry tasks that overstretch tendons, or have rest periods that are not sufficient to enable normal repair mechanisms to occur (Ashton-Miller, 1999, Ex. 26–414; Chaffin and Andersson, 1991, Ex. 26–420; Moore, 1992a, Ex. 26–985; Woo *et al.*, 1994, Ex. 26–596). Unfortunately, many jobs and tasks in manufacturing and other work settings associated with excessive hand force, machine paced or piece work, overtime, poor tool design, etc. have these associated risks. In addition, repetitive tendon loading may cause tendon deformation and eventual tissue failure at a lower limit during subsequent loading cycles (Goldstein *et al.*, 1987, Ex. 26–953; Moore, 1992a, Ex. 26–985; Thorson and Szabo, 1992, Ex. 26–1171). Compression and friction of tendons as they cross joints or move through tight compartments (*e.g.*, the carpal canal or first dorsal compartment of the wrist) may result in inflammation, degeneration, and metaplastic changes with symptoms and signs of tendon pathology (*e.g.*, stenosing tenosynovitis, tenosynovitis, tendinitis) (Ashton-Miller, 1999, Ex. 26–414; Azar *et al.*, 1984, Ex. 26–1031; Backman *et al.*, 1990, Ex. 26–251; Finkelstein, 1930, Ex. 26–266; Flint *et al.*, 1975, Ex. 26–268; Goldstein *et al.*, 1987, Ex. 26–953; Hart, Frank, and Bray, 1994, Ex. 26–551; Kilbom, 1994, Ex. 1352; Rais, 1961, Ex. 26–1166; Rathburn and McNab, 1970, Ex. 26–1376; Sampson *et al.*, 1991, Ex. 26–322; Uchiyama *et al.*, 1995, Ex. 26–339; Vogel, 1994, Ex. 26–593; Wilson and Goodship, 1994, Ex. 26–241).

Tendons and ligaments are connective tissues that connect either muscle to bone (tendons), or bone to bone (ligaments). Tendons and ligaments are relatively uncomplicated tissues, with a simple structure subject to a limited set of stresses: tensile forces from muscle contraction, shear forces from friction against obstructing anatomic structures, and compressive forces from entrapment. Injuries to the muscle and tendon unit are common in the upper extremity.

Tendon structure consists of parallel-oriented collagen bundles in a water-mucopolysaccharide matrix. In ligament, bundles are primarily parallel, with some bundles arranged in a non-parallel fashion. This results in different mechanical properties for these tissues, with more elasticity noted in ligamentous structures (Chaffin and Andersson, 1991, Ex. 26–420).

Tendons. Skeletal muscle, unlike tendon, is composed of non-parallel fibers. Therefore, as the muscle-tendon unit proceeds from muscle to tendon (myotendinous junction), intracellular contractile muscle proteins transition to extracellular collagen in the tendon, and the arrangement of collagen fibers becomes more parallel. Extensive infolding of fibers in the myotendinous junction increases the surface area of the muscle-tendon interface and decreases the stress from tensile loading in this area (Chaffin and Andersson, 1991, Ex. 26–420). The myotendinous junction then proceeds to a region called the aponeurosis, where tendon connective tissue predominates. Peritenon, a thin membranous sheath, separates the aponeurosis from the surrounding fascia.

Microscopically, the distal tendon consists of multiple bundles of collagen tissue surrounded by epitenon, endotenon, and peritenon membranes. The extracellular matrix of healthy tendon includes water, glycosaminoglycans, and glycoproteins. Blood vessels, lymphatics, and nerves may traverse the epitenon or endotenon layers. However, avascular regions are observed in healthy tendons, and it is presumed that these regions are nourished by diffusion. The distal tendon has a synovial sheath that produces lubricating fluid (synovial fluid). In the

hand, transverse ligaments called pulleys are present near the distal metacarpal and permit flexor tendons to flex the finger through a fibroosseous canal without bowing out.

The primary function of tendon is to transmit forces from muscle to bone. Accordingly, its principal injuries involve forces causing stretch, deformation, or inadequate recovery (*i.e.*, return to resting length), on the one hand, and frictional damage due to shear and extrinsic compression, on the other. The tendon is subject to both uniaxial tensile forces from muscles and transverse forces from anatomic pulleys, bursae, and extended range of motion. Tensile and transverse forces produce shear and influence tendon gliding. This phenomenon draws particular attention to awkward or extreme posture, particularly at the wrist (Armstrong *et al.*, 1984, Ex. 26–1293).

Pathophysiologically, four main types of non-acute tendon disorders have been suggested (Leadbetter, 1992, Ex. 26–157). Paratenonitis (tenosynovitis) is the inflammation of the peritenon. Signs and symptoms can include pain, swelling, warmth, and tenderness. Tendinosis involves intratendinous degeneration with fiber disorientation, scattered vascular ingrowth, occasional necrosis, and calcification; tendon nodularity may be noted, but swelling of the tendon sheath is absent. Paratenonitis may be observed with tendinosis. Corresponding signs of inflammation and nodularity are possible. Tendinitis (tendon strain or tear) can range from inflammation with acute hemorrhage and tear to inflammation with chronic degeneration. Clinical symptoms and signs relate to the contributions of inflammation vs. degeneration. This classification into four types, however, is not universally accepted.

To understand how tendons become diseased, one must understand tendon function and repair mechanisms. As muscles contract, tendons are subjected to mechanical loading and viscoelastic deformation. Tendons must have both tensile resistance to loading (to move attached bones) and elastic properties (to enable them to move around turns, as in the hand). When collagen bundles are placed under tension, they first elongate without significant increase in stress. With increased tension, they become stiffer in response to this further loading. If the load on these structures exceeds the elastic limit of the tissue (its ability to recoil to its original configuration), permanent changes occur (Ashton-Miller, 1999, Ex. 26–414; Moore, 1992a, Ex. 26–985; Chaffin and Andersson, 1991, Ex. 26–420). During subsequent loading of the damaged tendon, less stiffness is observed. The ultimate strength of normal tendon and ligament is about 50% of that of cortical bone (Frankel and Nordin, 1980, Ex. 26–1125), but structures that have exceeded the elastic limit fail at lower limits. In addition, if recovery time between contractions is too short, deformation can result in pathologic changes that decrease the tendon's ultimate strength (Thorson and Szabo, 1992, Ex. 26–1171; Goldstein *et al.*, 1987, Ex. 26–953).

Tendon exhibits additional viscoelastic properties of relaxation and creep. That is, when a tendon is subjected to prolonged elongation and loading, the magnitude of the tensile force will gradually decrease (relaxation) and the length of the tendon will gradually increase (creep) to a level of equilibrium (Chaffin and Andersson, 1991, Ex. 26–420; Moore, 1992a, Ex. 26–985; Woo *et al.*, 1994, Ex. 26–596). During repetitive loading, the tendon exhibits these properties and then recovers if there is sufficient recovery time. If the time interval between loadings does not permit restoration, then recovery can be incomplete, even if the elastic limit is not exceeded (Goldstein *et al.*, 1987, Ex. 26–953).

Tendons are also subject to perpendicularly oriented compressive loading. This is seen when tendons are loaded as they turn corners around pulleys or bony surfaces. Friction is generated at these locations as the tendon slides against adjacent surfaces, causing a shearing force. This is significant in the hand and wrist, as demonstrated by Goldstein *et al.* (1987, Ex. 26–953). The authors noted that higher levels of muscle tension are required to achieve a specific level of strength at the fingertip during non-neutral wrist postures, and that tendons are subject to greater shear stress with non-neutral wrist postures. Similarly, compressive force in the A1 pulley has been demonstrated to rise dramatically from the neutral posture (0 to 50 mm Hg) to full flexion (500 to 700 mm Hg) (Azar, Fleeger, and Cluver, 1984, Ex. 26–1031). Tendon friction is proportional to the axial tension of the tendon, the coefficient of friction between the tendon and its adjacent surface, and the angle of the tendon as it turns about a pulley (Uchiyama *et al.*, 1995, Ex. 26–339). Ashton-Miller, Ex. 26–414, suggests that this may be a cause of surface degeneration in tendons. Internal degeneration may be the result of friction-induced internal heat generation (Wilson and Goodship, 1994, Ex. 26–241). One study in exercising racehorses demonstrated that tendon core temperature in the superficial digital flexor tendon was 5.4 degrees above tendon surface temperature, enough to kill fibroblasts in vitro (Wilson and Goodship, 1994, Ex. 26–241).

Clinically, tendon compression in the hand may manifest as stenosing tenosynovitis. Initially, examination in patients with stenosing tenosynovitis may reveal impaired motion, tenderness, pain on resisted contraction or passive stretch, swelling, or crepitation. With time, swelling and thickening of the tendon may occur from fibril disruption, partial laceration, impairment of blood flow and diffusion of metabolites, and the localized repair process. Ultimately, this limits the normal smooth passage of the tendon through its fibroosseous canal. These chronic tissue changes are recognized as triggering. At surgery, findings may include tightness and thickening of the pulley, nodular fusiform tendon swelling, fibrocartilaginous metaplasia, or fraying of the tendon (Finkelstein, 1930, Ex. 26–266; Sampson *et al.*, 1991, Ex. 26–322)

These conceptualized patterns of tendon injury have practical clinical significance, relating to some of the most common MSDs encountered in clinical practice. Micro-tears and gross trauma to the tendon produce an acute inflammatory condition with regeneration and removal of tissue debris. As noted, when the tendon load is great and there is insufficient recovery time between deformations for the tendon to recover its resting length, viscous strain can exceed elastic strain (Goldstein *et al.*, 1987, Ex. 26–953), causing tendon deformation (Thorson and Szabo, 1992, Ex. 26–1171). These are the mechanisms most often involved in the common ''sprain.''

A different injury mechanism occurs when tendon and tendon sheaths are forced over hard anatomic surfaces, producing either an inflammatory tendinitis or a zone of avascularity (lack of blood flow) due to compression (Rathburn and McNab, 1970, Ex. 26–1376). This has been experimentally demonstrated by electrically stimulating muscles to contract, causing friction and tendinitis (Rais, 1961, Ex. 26–1166). Impaired circulation, hard tissue compression, and degenerative change are pertinent to rotator cuff injuries, where tendon insertions on the greater tuberosity of the humerus can be compressed under the coracoacromial arch. Muscle tension, itself, can also restrict circulation when the tendon's supply of arterial blood runs

through the contracted muscle, as is the case with the supraspinata (Herberts *et al.,* 1984, Ex. 26–51). Common rotator cuff diagnoses that fall short of surgical intervention often fall under these pathophysiologic mechanisms.

A more subtle friction-related injury is de Quervain's Syndrome, in which a narrowed first dorsal compartment juxtaposes crossed tenosynovium of the abductor pollicis longus and extensor pollicis brevis (Witt *et al.,* 1991, Ex. 26–242). Injury in the first dorsal compartment in de Quervain's Syndrome is actually a disorder of the retinaculum, a specialized ligamentous tissue acting as an anatomic pulley to prevent tendon bowstringing, and involves the fingers and the toes. ''Bowstringing'' refers to the tendency of a tendon, under tension, to assume the shortest distance between its proximal and distal insertion, unless it is tethered and damped. The disorder is a hypertrophy of this retinaculum. Tendon and ligament are elastic and will ''creep'' (*i.e.,* stretch) in response to tensile loading. Creeping involves progressive fiber recruitment and loss of the natural waviness of collagen fibers.

A diversity of clinical terms complicates the description of tendon injuries. As Waldron points out (1989, Ex. 26–509), the traditional peritendinitis crepitans, characterized by an edematous or swollen musculo-tendinous junction, is more limited than the variety of soft tissue pains that are currently described as tendinitis or tenosynovitis. In the older usage, tendinitis was an uncommon and severe condition in which the injured tissues were swollen and crackled under compression. Currently, ''tendinitis'' is used to describe a wide variety of soft tissue pain and is the most widely used term employed to characterize MSDs. Tendons have very different structures, depending on anatomic location and function, so as a general term for a diseased tendon, ''tendinitis'' groups together several different pathologies. In the case of epicondylitis, the insertional tears seen in young athletes playing racket sports have little in common with the non-inflammatory degeneration seen in older populations, whether or not work is implicated as a risk factor (Chaard *et al.,* 1994, Ex. 26–458). The frequent lack of connection between observed gross pathology and clinical or reported symptoms is another consideration. In autopsy series, the majority of cadavers have tears at the TFCC (triangulate fibro-cartilage complex) in the wrist or degeneration of the ECRB (extensor carpi radialis brevis) insertion at the elbow (Mooney and Poehling, 1991, Ex. 26–304; Cherniack, 1996, Ex. 26–258). However, the occurrence of perceptible symptoms is comparatively uncommon.

Tendons and ligaments also undergo significant modification where they turn corners or insert onto bone. Evidence exists that the tendon matrix is reformulated in response to mechanical forces, implying an active process of cell response. However, it has not been determined whether this reaction definitively alters the mechanical properties of the tendon, or what its role is in future injury. Experimental work with rabbit flexor digitorum profundus tendon compressed by adjacent calcaneum and talus (Flint *et al.,* 1975, Ex. 26–268) has demonstrated that fibrocartilagenous metaplasia occurs in response, and that after surgical translocation of the tendon, this will improve. The presence of sex hormone and neurotransmitter receptors in tendon tissues indicates that tissue responses are complex (Hart, Frank, and Bray, 1994, Ex. 26–551). This implies that tendon is affected by internal signals and is subject to regulation beyond stress and strain. The proinflammatory neurotransmitters substance P and calcitonin gene-related peptide are located in the nerve endings present in tendons and ligaments (Goldstein *et al.,* 1987, Ex. 26–953) and constitute a pathway for neurologically mediated tendon injury. The current notion of tendons and ligaments, which are structurally closely related, describes them as dynamic tissues subject to biomechanical strain and the effects of endocrine hormones and neurotransmitters. This suggests potentially complex patterns of injury and pain, and also of adaptation. Although a complete view of tendon function remains to be articulated, for now it seems clear that remodeling of tendons, inflammation, and the response to injury are mediated systemically as well as locally.

Additional experimental evidence relates to a more chronic or cumulative process through which tendon injury can evolve. Much is unknown about underlying pathophysiologic mechanisms in even such common mechanical tendon-and tenosynovium-related disorders as breakdown of the ECRB in lateral epicondylitis and tendinitis of the flexor digitorum in CTS. However, the provocation of a tissue response characterized by proinflammatory mediators in laboratory animals exposed to continuous motion (Backman *et al.,* 1990, Ex. 26–251) strongly suggests that biomechanical loading and stresses induce mechanical tissue injury and acquired micro-structural changes. Although this provides a useful direction, laboratory tendon loading experiments have not permitted a human threshold for repetitions causing tendon injury to be quantified.

Experience suggests that resolution of tendinitis can be surprisingly time-consuming. The reasons can be found in the pathophysiology of tendon repair. Following flexor tendon laceration, tendon healing follows three phases. Initially, inflammation is observed, with cells arising from the epitenon, endotenon, and peritendinous tissue. This stimulates migration and proliferation of fibroblasts and the removal of damaged tissue. The inflammatory phase ends long before tissue remodeling has been completed. Within the first week, collagen synthesis is initiated, though fiber orientation may be chaotic. By the fourth week, fibroblasts predominate and collagen content increases. Maturation of collagen and functional alignment occurs by the second month, with maximum functional restoration requiring exposure of the healing tendon to renewed loading. Exercise and movement are fundamental to the therapeutic process of an injured tendon. But premature exercise can be detrimental; movement of a deformed, devascularized, or inflamed tendon will provoke further injury and breakdown. Mechanical loading that results in a stiffer tendon development can provide structural integrity but a loss of mobility. Pain is an important indicator of either gross or microscopic abnormal tissue responses. In considering MSDs involving tendon and ligament it is especially important to differentiate between aggravation of an injury and exercise, which can be therapeutic. Exercise has proven to be an important component in the remodeling and strengthening of the ligaments of the rat knee (Frank, McDonald, and Shrive, 1997, Ex. 26–623). However, tendon and ligament adaptation and repair are inevitably slow processes'a knee injury can take up to 2 years to fully repair. Thus, although tendon, in particular, can effect a considerable but slow adaptational response to increased physical demand, a progressive increase in loading demands can easily exceed remodeling capacity, increasing the likelihood of re-injury. The slow natural rate of tendon and ligament repair also highlights the importance of prevention and early intervention. Established injuries can persist for weeks and months even after ergonomic review of the workplace and remediation.

In summary, clear evidence exists to support the conclusion that conditions often present at work can be pathogenic for some tendon disorders, as discussed above. Potential etiologies include mechanical disadvantage or tendon related to changes in joint position, changes in tensile and viscoeastic properties of tendon with excessive or repetitive loading, interference with normal repair mechanisms, and the effects of compression and friction leading to internal and external degeneration and inflammatory responses.

Ligaments. Work exposures may contribute to the development of ligament and joint disorders as the result of many pathogenic and physiologic mechanisms. Ligaments, like tendons, have viscoelastic properties that may be exceeded by repetitive loading or deformation, resulting in possible subsequent failure during lower levels of loading (Chaffin and Andersson, 1991, Ex. 26–420). On the one hand, ligamentous laxity has been demonstrated in the wrist after continuous exercise (Crisco *et al.,* 1997, Ex. 26–1373). This type of stress is commonly observed in highly repetitive work settings. On the other hand, immobilization may result in decreased ligamentous tensile strength (Woo *et al.,* 1987, Ex. 26–243). The significance of this finding in workers who perform prolonged, sedentary work merits further investigation.

Although tendon and ligament have many structural similarities, they also have important differences. Ligament structure consists of type I and type III collagen with elastin and glycosaminoglycans. Ligamentous structures are somewhat more elastic than tendon, in part because of the occurrence of non-parallel fibers. As in tendon, there are length and velocity tension relationships, and relaxation and creep are noted (Chaffin and Andersson, 1991, Ex. 26–420). The ability of ligaments to adapt to changes in physiologic loading has been studied in the rabbit medial collateral ligament. After 9 weeks of immobilization, a 50% decline in tensile strength was noted (Woo *et al.,* 1987, Ex. 26–243). With remobilization, stiffness improved, but after 9 weeks was still 20% below initial values. Viscoelastic changes have been reported with repetitive loading, with a 30% increase in wrist laxity in subjects performing 1 hour of exercise. After 24 hours, tissue laxity had returned to baseline (Crisco *et al.,* 1997, Ex. 26–1373). Ligament healing and remodeling is, unfortunately, rather slow and limited. After injury, a vascular response is rather prolonged, and can last for several months (Bray *et al.,* 1996, Ex. 26–773). With aging, a decrease in elastic stiffness and failure can occur at lower loads, as demonstrated in a study comparing tissue samples from old (mean age 76 years) and young (mean age 35 years) subjects (Woo *et al.,* 1991, Ex. 26–244).

Joint hypermobility, the familiar double-jointedness, appears to be more common in women than in men (Bridges *et al.,* 1992, Ex. 26–1312), and appears to have a strong genetic basis (Child, 1986, Ex. 26–358). It is more an anthropometric factor, or effect modifier, than a predisposition to disease. That is, hypermobility is not an intrinsically morbid condition, but it can increase musculo-tendinous loading and effort. It has been recognized as a risk factor for musculotendinous injury in hand-intensive tasks, presumably because of the co-contractive effort required to stabilize small joints in the hand (Pascarelli *et al.,* 1993, Ex. 26–1164). Hyper-mobility means that opposing muscle groups must be simultaneously and antagonistically contracted to maintain the position of a finger or a wrist against resistance. There is considerable speculation that hormones, as well as mechanical stresses, may influence knee and other tendon and ligament injuries in women. Although it is premature to ascribe these factors to the risk of developing a work-related knee injury, it is important to recognize that ligamentous laxity can usually be accommodated through changes in work technique and job design.

Ligamentous laxity is also acquired in the course of continuous work. A 30% increase in wrist laxity (due to visco-elastic stretching) has been observed after 1 hour of continuous exercise (Crisco *et al.,* 1997, Ex. 26–1373). There is a return to normal length and function within 4 hours. This observation highlights the point that maintenance of ligamentous function requires periods of rest and disuse.

c. Nerve. Work-related nerve disorders include compression, entrapment, and vibration-induced and toxic neuropathies. It is the first two that are within the scope of this document. Compression most commonly occurs adjacent to joints or as nerves pass through muscle or connective tissue. This may result in mechanical deformation of nerves, perineural edema, nerve ischemia, and inflammation with secondary nerve damage and delayed conduction (Feldman *et al.,* 1983, Ex. 26–949; Gelberman *et al.,* 1983, Ex. 26–465; Lundborg and Dahlin, 1994, Ex. 26–561; Moore, 1992b, Ex. 26–984; Rydevik *et al.,* 1989, Ex. 26–198; Szabo *et al.,* 1983, Ex. 26–333). Examples of this include carpal tunnel syndrome, cubital tunnel syndrome, entrapment at Guyon's canal, and tarsal tunnel syndrome (Bozentka, 1998, Ex. 26–82; Delisa and Saeed, 1983, Ex. 26–364; Feldman *et al.,* 1983, Ex. 26–949; Moore, 1992b, Ex. 26–984; Terzis and Noah, 1994, Ex. 26–587). External compression with impairment of nerve function may occur from contact stress between body parts and hard work surfaces or sharp edges (*e.g.,* carpal tunnel syndrome, cubital tunnel syndrome) (Feldman *et al.,* 1983, Ex. 26–949; Hoffman and Hoffman, 1985, Ex. 26–141). Alternatively, internal compression may occur from increased compartmental pressures or from contact against bones, tendons, or ligaments (*e.g.,* cubital tunnel syndrome, carpal tunnel syndrome) (Bozentka, 1998, Ex. 26–82; Feldman *et al.,* 1983, Ex. 26–949; Moore, 1992b, Ex. 26–984; Skie *et al.,* 1990, Ex. 26–328). At times, workers may experience anatomic and tissue changes with multiple sites of nerve compression that cause greater damage than would be experienced with a single site of compression (''double crush syndrome'') (Lundborg and Dahlin, 1994, Ex. 26–949; Mackinnon, 1992, Ex. 26–646; Novak and Mackinnon, 1998, Ex. 26–1310). Furthermore, whole-body vibration transmitted by vehicles or segmental vibration from hand tool use may damage nerves directly or indirectly because of ischemia or adjacent tissue changes (Hjortsberg *et al.,* 1989, Ex. 26–1131; McLain and Weinstein, 1994, Ex. 26–1347; NIOSH, 1989, Ex. 26–392; Takeushi *et al.,* 1986, Ex. 26–681; Rempel *et al.,* 1998, Ex. 26–444).

Peripheral nerve is composed of a nerve cell body (motor or sensory) and an axon, which extends to the periphery. An axon with its sheath constitutes a nerve fiber. Myelinated fibers are surrounded by single layers of Schwann cells arranged in a longitudinal manner along the nerve. Spaces on myelinated nerves created by adjacent Schwann cells are called nodes of Ranvier. Bundles of nerve fibers, called fascicles, are wrapped by perineurium and embedded with microvasculature in epineural tissue. The amount of epineural tissue and the presence or absence of myelination depends on the location and purpose of the nerve. The largest myelinated fibers (Group A) have the highest conduction velocity. Group B fibers are myelinated autonomic and preganglionic fibers. The thinnest, non-myelinated fibers have the lowest conduction velocity and

make up the visceral and somatic afferent pain Group C fibers.

Substances required for membrane integrity are synthesized in the nerve cell body and transported to the periphery, while disposal of waste materials and transport of trophic and tropic factors both involve transport from the periphery to the nerve cell body (Lundborg and Dahlin, 1994, Ex. 26–561). Both propagation of impulses and transportation of materials require a sufficient energy supply and vasculature. Depending upon location, peripheral nerves are subject to variable amounts of gliding or excursion in response to muscle, tendon, and joint movement (Bozentka, 1998, Ex. 26–82; Chaffin and Andersson 1991, Ex. 26–420; Novak and Mackinnon, 1998, Ex. 26–1310; Rempel, Dahlin, and Lundborg, 1998, Ex. 26–444).

There are several mechanisms by which peripheral nerves are either injured directly or contribute secondarily to pain and dysfunction. Nerve tissue plays a predominant role in transmitting information on the extent of tissue damage and in establishing the CNS link producing sensations of pain. Movement disorders and dystonias, which produce chaotic or uncontrolled patterns of hand movement or cramps, also involve patterns of abnormal nerve transmission, but here the problem has more to do with function and control than pain. Nerve tissue can also be directly injured, producing characteristic symptom patterns.

The most widely recognized lesions of peripheral nerves associated with repetitive work and chronic overuse are the entrapment and compression neuropathies. Mechanical pressure on a peripheral nerve, if severe enough, causes a block or delay in the conduction of nerve impulses, a decline in sensory function, and paresthesias (''pins and needles''). Because defects in the conduction of nerve impulses can be assessed by electrophysiology (Wilbourn and Lederman, 1984, Ex. 26–1409) or by shifts in thresholds of perception (Lundborg *et al.*, 1987, Ex. 26–645), nerve entrapments have traditionally been the most effectively studied MSDs. The notion of nerve entrapment implies that external pressure or resistance on a peripheral nerve restricts free nerve movement or impinges on nerve contents (Lundborg, 1988, Ex. 26–1145). This pressure or resistance can be caused by external compression through soft tissue swelling by a fracture or callus, or by swelling or scarring of the nerve tissues themselves. The necessity for peripheral nerves to move during musculoskeletal activity is often underappreciated, with ulnar nerve range at the elbow approaching 1.5 cm and median nerve mobility being 1.0 cm at the wrist (Millesi *et al.*, 1990, Ex. 26–567). In the upper extremity, areas of potential nerve compression are most frequently situated in the vicinity of joints. The two most common upper-extremity disorders are CTS at the wrist and cubital tunnel syndrome at the elbow. In the low back, degenerative disease and bony compression of nerve roots is the most common cause of radicular pain patterns (Deyo *et al.*, 1990, Ex. 26–106).

The histopathology of human compressive neuropathy has not been well studied, because surgical management does not provide pathological specimens. However, findings from animal experiments appear to correlate with the limited findings from human specimens where nerve was resected or from an autopsy on an individual with compressive neuropathy (Novak and Mackinnon, 1994, Ex. 26–1310; Mackinnon *et al.*, 1986, Ex. 26–1321; Rempel, Dahlin, and Lundborg, 1998, Ex. 26–444; Terzis and Noah, 1994, Ex. 26–587). After compression of nerve, changes in the blood-nerve barrier develop and are followed by subperineurial edema and thickening of both perineurial and epineural layers (Lundborg and Dahlin, 1994, Ex. 26–561 ; Novak and Mackinnon, 1998, Ex. 26–1310; Rempel, Dahlin, and Lundborg, 1999, Ex. 26–444; Terzis and Noah, 1994, Ex. 26–587). After intraneural fibrosis, myelin thinning results, with fibers at the periphery of the nerve affected first. If compression continues, segmental demyelination progresses to more diffuse demyelination and, finally, axonal degeneration occurs (Mackinnon and Dellon, 1988, Ex. 26–296; Mackinnon *et al.*, 1984, 1985, Exs. 26–648 and 26–649).

Histopathologic changes are dependent on the force and duration of compression, as well as the characteristics of the nerve. Changes can also vary among different fascicles within the nerve (Mackinnon, 1992, Ex. 26–646). Nerves composed of large amounts of connective tissue with relatively few fascicles may be less susceptible to injury (Dickson and Wright, 1984, Ex. 26–1298; Lundborg, 1988, Ex. 26–1145). The nearer nerve fascicles are to the site of compression, the sooner pathologic changes will occur.

Laboratory observations appear to support these conclusions. In a study of canine extensor digitorum brevis muscle, Hargens *et al.* (1979, Ex. 26–135) created a compartment syndrome by infusing plasma. As pressure rose, the amplitude of the action potential declined until complete nerve block developed at 2 hours at pressures of 80 to 120 mm Hg. Histopathological evidence of axonal degeneration was noted after 3 weeks. Graded external compression of rabbit tibial nerve demonstrated complete interference with epineural venular, arteriolar, and intrafascicular capillary flow at pressures from 60 mm Hg to 80 mm Hg (Rydevik *et al.*, 1981, Ex. 26–321). The neural ischemia may then cause endoneurial edema, with further rises in intraneural pressure.

As nerves are stretched over another anatomic structure, mechanical deformation can occur with microruptures, abnormal function (ischemia and decreased nerve conduction) and scarring (Armstrong, 1983, Ex. 26–927). In addition, there can be an incompatibility between the anatomic space available for the nerve and the volume and pressure of the space (Lundborg, 1988, Ex. 26–1145). For example, in cubital tunnel syndrome, repeated flexion results in stretch and friction of the ulnar nerve (Harter, 1989, Ex. 26–958). This can be compounded by elevations in the pressure in the cubital tunnel that have been observed with elbow flexion (Pechan and Julis, 1975, Ex. 26–575). Elbow flexion also places the ulnar nerve in a more superficial position, where it can be damaged by leaning the elbow on a work surface.

Because it is the most common nerve entrapment disorder of the upper extremity and because it is easily studied, CTS has become the benchmark nerve compression disorder (Szabo and Gelberman, 1987, Ex. 26–1013). In CTS, postural extremes can cause significant increases in mean intracarpal pressures from 2.5 to 30 mm Hg in normal subjects, and from 32 to 94 (flexion) or 110 (extension) mm Hg in patients with CTS (Gelberman *et al.*, 1981, Ex. 26–1127; Szabo and Chidley, 1989, Ex. 26–1168). Similarly, pressures can rise with exposure of flexor tendons to high forces (Smith, Sonstegard, and Anderson, 1977, Ex. 26–1006), or repetitive hand/wrist motions (Gelberman *et al.*, 1981, Ex. 26–1127; Szabo and Gelberman, 1987, Ex. 26–1013). Within 1 hour, elevated carpal tunnel pressures can result in impaired conduction and median nerve sensory function (Gelberman *et al.*, 1981, Ex. 26–1127; Lundborg, 1988, Ex. 26–1145). Even transient increases in intracarpal pressure can produce slowed nerve conduction and altered sensory function of the hand (Lundborg *et al.*, 1982, Ex. 26–979). These types of

pressure can be induced by prolonged isotonic or isometric contractions of wrist and digital flexors (Werner, Elmquist, and Ohlin, 1983, Ex. 26–1025). Studies of intracarpal pressure in these more exaggerated or non-neutral positions have had consistent results, demonstrating large increases in pressure when the wrist is forcefully stressed, particularly in hyperextension (Rempel *et al.*, 1994, Ex. 26–1151; Werner *et al.*, 1994, Ex. 26–237). Relatively low fingertip loads (5 to 15 N) raise carpal tunnel pressures by 4 to 6.6 kPa (Rempel *et al.*, 1997, Ex. 26–889). Classic studies in the meatpacking industry (Masear, Hayes, and Hyde, 1986, Ex. 26–983) and in the automobile industry (Silverstein, Fine, and Armstrong, 1987, Ex. 26–34) have shown a consistent pattern of forceful wrist exertions and nerve compression syndromes. This same pattern of risks is evidenced in the so-called pinch grip, leading to innovations in tool handle design (Tichauer, 1978, Ex. 26–446). Use of modifications tend to involve the full palm rather than the fingers alone.

Because of the strong association of CTS with repetitive and forceful work and awkward postures (Silverstein, Fine, and Armstrong, 1987, Ex. 26–34), there has been particular attention to the process by which joint deviation and loading and repetitive muscle contraction can raise pressure at an anatomic canal. In the upper extremity, fibrotic changes in the radial and ulnar bursae and at the carpal tunnel have been located consistently. These changes potentially produce compressive stresses on the median, ulnar, and radial nerves from bone and retinaculum (Armstrong *et al.*, 1984, Ex. 26–1293).

The transition from acute compression injury to a chronic nerve entrapment condition involves an extension of these pathophysiologic models. However, Mackinnon *et al.* (1984, Ex. 26–648) have presented a histologic model showing the gradual transition from a recoverable nerve compression injury, in which there is swelling and thickening of the connective tissue lining bundles of nerve fibers, to demyelination of the nerve and nerve fibrosis, in which there are often irreversible changes to the nerve. This has been extended to a model of CTS (Mackinnon and Novak, 1997, Ex. 26–1309).

Novak and Mackinnon (1998, Ex. 26–1310) suggest that many patients with diffuse upper-extremity symptoms may experience problems from multiple levels or sites of nerve compression and concomitant muscle imbalance. These observations come from the often surprising clinical evidence that symptomatic patients often express signs at multiple sites of potential compression. This so-called ''double crush'' syndrome (Hurst *et al.*, 1985, Ex. 26–965) can be a consequence of degenerative cervical spine disease or acquired postural torsion at the brachial plexus (Mackinnon and Novak, 1997, Ex. 26–1309). In the ''double crush'' syndrome, there is compression at the carpal tunnel as well.

The concept of ''double'' or ''multiple crush syndromes'' is a controversial subject. In 1973, Upton and McComas first proposed that a proximal site of nerve compression, such as a cervical disc herniation, could make a distal nerve more susceptible to injury. Other potential scenarios could include ulnar nerve entrapment at the brachial plexus and cubital tunnel, or at the cubital tunnel and Guyon's canal. Mackinnon (1992, Ex. 26–646) and Dellon and Mackinnon (1991, Ex. 26–616) have further describe the concept. These observations can be significant in situations where work postures place muscles in shortened positions. For example, workers who perform tasks requiring prolonged or resisted pronation may develop pronator muscle shortening that compresses the median nerve in the forearm when the forearm is placed in supination. Alternatively, prolonged and static work postures that result in pectoralis minor or scalene muscle tightness can compress the brachial plexus. Alterations in axoplasmic flow and transport of neutrophic substances has been proposed as the mechanism of this injury. Dellon and Mackinnon (1991, Ex. 26–616) devised an experimental animal study to evaluate these phenomena. The authors banded either sciatic nerve, posterior tibial nerve, or both nerves in rat subjects. The group of rats with double banding demonstrated significantly worse mean amplitudes of the compound action potential than either group of single-banded rats. In theory, metabolic abnormalities (*e.g.*, diabetes, alcoholic neuropathy, collagen vascular disease) could weaken a nerve and make it more susceptible to injury from less significant levels of compression. In the case of diabetes, a recent article by S.E. MacKinnon (1992, Ex. 26–646) describes rodent and primate models of diabetes with superimposed nerve compression. With alcohol, it is biologically plausible, although not specifically documented, that a ''sick'' neuron resulting from alcoholism could similarly render a nerve metabolically damaged and therefore more susceptible to injury from compression at a distal site.

A related observation is that persistent stretching of a nerve over an anatomic landmark, such as the ulnar nerve at the medial epicondyle of the elbow, can produce nerve trauma and inflammation (Harter, 1989, Ex. 26–958). The notion that micro-ruptures produce micro-anatomic injury and fibrosis of the epineurium (connective tissue lining the nerve) has been offered as a general model for CTS (Armstrong *et al.*, 1993, Ex. 26–1110). This model has its analogue in the epineural fibrosis that can be a consequence of nerve release surgery.

It is important to recognize that CTS is not responsible for all cases of numbness and tingling in the fingers that occur in demanding work settings. Furthermore, there is no ''gold standard'' for diagnosis, and the presence of even classical symptoms does not necessarily mean that surgery is required. There is a high level of reversibility in CTS, and job modification can be enough to eliminate symptoms without aggressive individual therapy. Moreover, without job modification, surgery may only delay a recurrence. Even for this most accessible MSD, modest changes in diagnostic criteria—for example, whether symptoms and signs are weighted or full reliance is placed on the nerve conduction study—can alter the case rate by as much as 50% (Katz *et al.*, 1991, Ex. 26–151; Moore, 1991, Ex. 26–1335, Cherniack *et al.*, 1996, Ex. 26–258).

Other work-induced causes of peripheral nerve injury, such as hand-arm vibration, can induce small fiber nerve injury that is unrelated to entrapment or compression (see Section D.3). The result, however, is a similar pattern of symptoms. Even when the pattern of nerve injury distinctly implicates a focal site of compression, there is no automatic requirement for surgical decompression. It is also important to recognize that in the setting of low-back pain, even when symptoms radiate to the lower extremity along a nerve dermatome, fixed nerve root lesions and the correlated need for decompression are relatively rare (Andersson and McNeill, 1989, Ex. 26–413). The same is probably true for CTS, although the proportion of surgical cases for CTS remains comparatively high.

Although most work-related peripheral entrapment disorders affect myelinated nerve fibers, there are other nerve tissue components that are at risk. Mechanoreceptors in the glabrous pads of the digits are intrinsic to touch and spatial discrimination (Vallbo and Johansson, 1984, Ex. 26–

717). Their quantitative function has been effectively assessed through the testing of vibrotactile thresholds (Brammer *et al.*, 1987, Ex. 26–935; Verrillo and Capraro, 1975, Ex. 26–591). Individual mechanoreceptors, such as Pacinian corpuscles, which measure acceleration as a sensation of touch, respond to particular frequencies of vibration. This principle is useful in establishing thresholds of response and function for individual mechanoreceptor populations. Mechanoreceptor injury is a well-recognized consequence of exposure to hand-arm vibration, and dysfunction documented in objective tests has correlated with decrements in hand performance and sensitivity (Virokannas, 1992, Ex. 26–1355). Quantitative sensory dysfunction consistent with mechanoreceptor injury has also been observed in manual workers unexposed to vibration, but for whom energy transfer still occurs in the form of shock and impact (Flodmark and Lundborg, 1997, Ex. 26–370).

There are several proposed mechanisms for the development of lumbar nerve root pain, including mechanical deformation, compression, ischemia, and inflammatory mediators. It appears that the spinal nerve root may be more susceptible to compression than peripheral nerves (Olmarker and Rydevik, 1991, Ex. 26–190). In an in-vivo experiment compressing the porcine cauda equina (Olmarker, Holm, and Rydevik, 1990, Ex. 26–518; Olmarker, Rydevik, and Holm, 1989, Ex. 26–191; Olmarker *et al.*, 1989, Ex. 26–311), venous flow was observed to cease at relatively low pressures (5 to 10 mm Hg), resulting in retrograde stasis of capillaries and impaired nutrient transport (Rydevik *et al.*, 1990, Ex. 26–197). Changes in the permeability of the spinal nerve root endoneurial capillaries, intraneural edema, increased endoneurial fluid pressure, and impaired nutrition of the nerve roots have been described by others as resulting from compression (Low and Dyck, 1977, Ex. 26–482; Low, Dyck, and Schmeizer, 1982, Ex. 26–385; Lundborg, Myers, and Powell, 1983, Ex. 26–162; Myers *et al.*, 1982, Ex. 26–308; Olmarker, Rydevik, and Holm, 1989a, Ex. 26–191; Rydevik, Myers, and Powell, 1989, Ex. 26–198).

Inflammatory mediators have also been implicated in the etiology for low-back pain, and histopathologic signs of inflammation have been observed in compressed nerve roots (Bobechko and Hirsch, 1965, Ex. 26–252; Diamant, Karlsson, and Nachemson, 1968, Ex. 26–261; Marshall, Trethewie, and Curtain, 1977, Ex. 26–483; Marshall and Trethewie, 1973, Ex. 26–564; Nachemson, 1969, Ex. 26–742). Proposed mediators include lactic acid, pH, substance P, bradykinin, cytokines, prostaglandins, and carrageenan, among others.

In recent years there has been a growing recognition of pain syndromes maintained by the sympathetic nervous system. These sympathetically maintained pain syndromes (SMPSs), of which reflex sympathetic dystrophy (RSD) is the best known, are characterized by pain and swelling, usually of the hands or feet, and vascular dysfunction (Roberts, 1986, Ex. 26–402; Kozin, 1994, Ex. 26–556). Traumatic origins are common, particularly following fracture to the hand, but there is evidence of a more widespread occurrence, in the setting of CTS, for example. This broader definition of SMPS appears to have substantial relevance to chronic soft tissue injuries, such as MSDs, associated with the workplace.

The evidence reviewed supports the conclusion that work conditions can be pathogenic for some nerve disorders. Mechanisms include external or internal nerve compression or mechanical deformation with subperineurial edema, altered metabolic nerve activity, demyelination, and axonal degeneration.

d. Vasculature. The ability of muscles, tendons, ligaments and cartilage to perform work and permit repair is dependent upon adequate blood flow, tissue oxygenation, and transmission of nutrients and metabolic end products. Therefore, when the performance of work tasks results in exposure to external or internal factors that impair normal tissue blood flow, tissue damage can occur and result in the development of MSDs. Mechanisms of injury may include tissue hypoxia from elevations in intramuscular pressure associated with forceful work or postural task requirements (Armstrong *et al.*, 1993, Ex. 26–1110; Herberts *et al.*, 1984, Ex. 26–51; Sjogaard and Sjogaard, 1998, Ex. 26–1322), vascular occlusion from direct pressure to anatomic structures (Duncan, 1996, Ex. 26–366; Kleinert and Volianitis, 1965, Ex. 26–380; Nilsson, Burstrom, and Hagberg, 1989, Ex. 26–693; Wheatley and Marx, 1996, Ex. 26–693), and vibration-induced vasospasm or impairment of microcirculation from hand tool use or whole-body vibration (Hirano *et al.*, 1988, Ex. 26–140; Kaji *et al.*, 1993, Ex. 26–854; NIOSH, 1989, Ex. 26–392). Thus it appears that vascular changes resulting from work exposures may contribute to the development or manifestation of MSDs.

The circulatory system is a major target of acquired morbidity for general health. However, while conditions such as atherosclerosis and smoking-related endothelial dysfunction can compromise neuromuscular function, their etiology does not evolve out of the workplace. Ischemia due to arteriosclerosis is an important component of muscle pain and dysfunction, but it is not a primary acquired work-related disorder. Ischemia caused by static contraction and transmural pressure from muscles and bone across arteries is work-related, and is usually reversible. There are distinct vaso-occlusive and vasospastic disorders of the hand that have a singular work-related etiology.

Arterial occlusive disease, expressed as either Raynaud's phenomenon or digital pain, has been described in a variety of hand-intensive tasks (Schatz, 1963, Ex. 26–200). Palmar and digital artery occlusion that is work-induced is usually due to traumatic ulnar artery occlusion, the so-called hypothenar syndrome or ulnar hammer syndrome (Wheatley and Marx, 1996, Ex. 26–693; Duncan, 1996, Ex. 26–366). The general mechanism causing thrombotic emboli in the palm and fingers is blunt trauma, caused by using the hand as a percussive object or by aggressively twisting hard objects (Pineda *et al.*, 1985, Ex. 26–493; Kreitner *et al.*, 1996, Ex. 26–557). The disorder has also been associated, albeit uncommonly, with the use of hand-held pneumatic tools (Kaji *et al.*, 1993, Ex. 26–854). The usual mechanism is ascribed to trauma and abrupt injury of the endothelium (the blood vessel lining), with the ulnar artery being bludgeoned against the hook of the hamate (Benedict, Chang, and McCready, 1974, Ex. 26–352). Contractions around the ulnar artery due to an anatomic muscle sling or anomalous hypothenar muscle has also been described (Benedict, Chang, and McCready, 1974, Ex. 26–352). Physiologically, the lesion is the consequence of thrombi, or small clots, that lodge in smaller or more peripheral vessels. This can occur because of pressure, the blockage of blood flow, and stasis-related clot formation. It is also hypothesized that shear forces injure the endothelium and expose the underlying tissues, the vascular intima, to injury. The repair mechanism leads to clot formation.

The most common vasospastic disorder associated with workplace exposure is occupational Raynaud's or vibration-induced white finger (VWF). In the field of hand-arm vibration, exposure measurement and specialized disease testing have produced highly evolved, methodologically

detailed, and technically sophisticated approaches that have few equivalents in the occupational health literature, and none in the literature on soft tissue injury. Because vibration is a complex physical factor, lending itself to quantification and modeling, and because it produces distinct and reproducible effects on vessels and nerves, there are parallels to noise in the formality of measurement methodology. VWF is largely associated with hand-held oscillating pneumatic tools, such as metal grinders and pneumatic drills. It is also associated with chain saws and with powered tools causing repetitive impact, such as riveters and impact wrenches. The mechanisms producing Raynaud's in the setting of hand-arm vibration are not fully understood. However, there is evidence for a sympathetically mediated constriction of small arteries in the hand, interrupting cutaneous blood flow. There is also evidence of impaired dilatation of larger arteries. Section D.3.b presents a more complete discussion of hand-arm vibration.

Vibration can also diminish the blood flow to the intervertebral disc. This has been demonstrated by Hirano *et al.* (1988, Ex. 26–140) in the rabbit intervertebral disc exposed to in-vivo vibration. Unfortunately, the lumbar intervertebral disc is avascular, and its nutritional supply comes from diffusion through blood vessels surrounding the annulus fibrosus and under the hyaline end plate cartilage. Diminished blood flow to the cartilage end plate would limit the ability of the disc to maintain the degree of hydration necessary to provide support for the lumbar spine during loading. In the hand, direct pressure over the hypothenar eminence can also occlude the ulnar artery and result in hypothenar hammer syndrome (Conn, Bergan, and Bell, 1970, Ex. 26–821; Kleinert and Volianitis, 1965, Ex. 26–380; Nilsson, Burstrom, and Hagberg, 1989, Ex. 26–1148). Thus, it appears that vascular changes resulting from work exposures may contribute to the development or manifestation of MSDs.

Extrinsic ischemic compression, while not an intrinsic disease of blood vessels, is also considered here to complete the discussion of vascular responses to work exposures. The ability of muscles, tendons, ligaments, and cartilage to perform work and permit repair depends on adequate blood flow, tissue oxygenation, and transmission of nutrients and metabolic end products. When external or internal factors impair normal tissue blood flow, tissue damage can occur and result in the development of MSDs. As discussed, elevations in intramuscular pressure with forceful exertion, confinement from bony structures, or tight fascial compartments can contribute to the onset of work-related MSDs as a result of tissue hypoxia (Armstrong *et al.,* 1993, Ex. 26–1110; Sjogaard and Sogaard, 1998, Ex. 26–1322). For example, work tasks that require shoulder abduction and/or elevation to perform activities at or above shoulder height can decrease blood flow to the hypovascular portion of the supraspinatus tendon (Herberts *et al.,* 1984, Ex. 26–51). A decrease in blood flow to the trapezius muscle has also been observed in assembly workers with localized chronic myalgia related to static loading (Larsson *et al.,* 1990, Ex. 26–1332).

e. Synovial Joints and Hyaline Cartilage. Work exposures may contribute to the development of joint disorders for many reasons. Joint cartilage matrix metabolism may be disturbed and inflammatory and chemical mediators stimulated by joint trauma or repetitive loading (Allan, 1998, Ex. 26–1316; Howell, 1989, Ex. 26–1308; Radin *et al.,* 1994, Ex. 26–578). Experimental animal studies have documented the loss of proteoglycans, fibroblast synthesis of inflammatory mediators, and the development of osteoarthritis from repetitive tissue loading (Allan, 1998, Ex. 26–1316; Farkas, 1987, Ex. 26–463; Poole, 1986, Ex. 26–1316; Vasan, 1983, Ex. 26–590). With inadequate repair, cartilage thinning and hypertrophic remodeling may lead to osteoarthritis (Chaffin and Andersson, 1991, Ex. 26–420; Radin, 1976, Ex. 26–663; Radin *et al.,* 1976, 1994, Exs. 26–443 and 26–578). Repetitive or prolonged stair or ladder climbing, kneeling or squatting, standing, carrying heavy loads, and jumping are all work tasks that may be associated with lower-extremity joint loading. This is explored further in the sections on epidemiology and pathogenesis of lower-extremity disorders. Recurrent microtrauma associated with the pinching mechanism, highly intensive hand tasks requiring dexterity during assembly work or food preparation, and pneumatic tool use have all been observed to associated with upper-extremity joint loading and the development of upper-extremity osteoarthritis (Bovenzi *et al.,* 1987, Ex. 26–605; Fam and Kolin, 1986, Ex. 26–1123; Felson, 1994b, Ex. 26–543; Nakamura *et al.,* 1993, Ex. 26–1314).

A synovial joint consists of bone ends covered by hyaline articular cartilage and separated by a synovial-fluid-filled joint cavity. A synovial membrane and capsule cover the joint. The joint capsule contains dense connective tissue and is attached to the distal ends of the articulating structures. It is innervated by sensory nerves that provide proprioceptive feedback and the sensation of pain. The normal synovium consists of one to three layers of cells. Type A synoviocytes are derived from monocytes and behave as phagocytes for joint space debris. Type B synoviocytes produce glucosaminoglycans for joint lubrication and enzymes in response to inflammatory stimuli. Cytokines secreted by both cells help to regulate the structural repair process after injury or antigenic stimulation (Allan, 1998, Ex. 26–1316).

Synovium has a rich vascular supply. It secretes synovial fluid and permits the transport of oxygen, carbon dioxide, nutrients, waste products, and immunologic cells to the joint. Trauma and inflammation impair the synovial microcirculation and transport of these substances across the joint.

There are three zones or layers of the articular cartilage. In the superficial zone adjacent to the joint cavity, collagen fibers are parallel to the articular surface. This orientation becomes more random in the middle zone. At the deep zone adjacent to the subchondral bone, fibers are mostly perpendicular because they anchor to the underlying bone (Allan, 1998, Ex. 26–1316; Mow, Lai, and Rodler, 1974, Ex. 26–653).

Collagen fibers are stable in the articular cartilage until degraded by age or disease, but proteoglycans are continuously synthesized by the chondrocytes (Allan, 1998, Ex. 26–1316). The proteoglycan matrix is hydrophilic, and osmotic pressure is resisted by tension in the collagen fibers in the unloaded joint. Once osmotic pressure is exceeded from external joint loading, water is squeezed out of the cartilage and the cartilage is flattened. Loaded, the articular cartilage undergoes elastic deformation followed by gradual creep. With unloading, the articular cartilage undergoes an initial elastic recoil followed by gradual recovery of its unloaded characteristics (Chaffin and Andersson, 1991, Ex. 26–420). Some joints, such as the knee, also contain fibrocartilage discs (menisci) to help protect the articular cartilage and distribute load forces.

It is clear that significant joint trauma can initiate hypertrophic remodeling, usually at sites of synovial

membrane and ligament attachment. The result is secondary cartilage breakdown (Howell, 1989, Ex. 26–1308). Unfortunately, cartilage has a limited vascular supply and ability to heal itself. With damage to subchondral tissues, there is reactive ossification and secondary cartilage thinning (Radin *et al.,* 1976, 1994, Exs. 26–443 and 26–578). After cartilage deteriorates, bone becomes subject to increased stress from loading, and reactive bone deposition occurs, resulting in sclerosis, spurring, or bone cysts noted in osteoarthritis. As the joint spaces narrow, the joint becomes more susceptible to further mechanical damage, inflammation, and scarring

Mechanical stresses associated with certain tasks that exceed the limits of tissue tolerance can either cause degenerative joint disease and/or accelerate the normal degenerative process that occurs with aging. They can also interact to hasten other forms of secondary osteoarthritis, including cases that occur after trauma or infection, and congenital, developmental, or anatomic abnormalities. For example, repetitive joint loading can impair cartilage matrix metabolism and disturb the repair processes (Allan, 1998, Ex. 26–1316; Radin *et al.,* 1994, Ex. 26–578). Studies of repetitive loading in dogs after 8 months of treadmill exercise have demonstrated a loss in proteoglycan similar to findings in models of osteoarthritis (Poole, 1986, Ex.26–1316; Vasan, 1983, Ex. 26–590). Rabbits subjected to 8 weeks of repetitive loading on the tibia show severe osteoarthritis after 24 weeks (Farkas *et al.,* 1987, Ex. 26–463). In-vitro fibroblast studies have also shown that repetitive motion can stimulate the synthesis of inflammatory mediators, including prostaglandins (Allan, 1998, Ex. 26–1316).

Degenerative joint disease can occur even after relatively low loads on joints if the forces are applied impulsively and repetitively (Radin and Paul, 1971, Ex. 26–496). This may occur because loads that are applied too rapidly to permit normal cartilage fluid movement could result in microscopic injury to the matrix (Radin *et al.,* 1994, Ex. 26–578). Loss of proteoglycans and cartilage fibrillation is also noted in this setting (Radin *et al.,* 1976, Ex. 26–443). Allan (1998, Ex. 26–1316) suggests that several joint interactions involved with repetitive loading may contribute to pathology. Since joints involve many structures, including tendon, muscle, nerve, and bone, damage to one structure may occur although the recovery cycle of another structure was not exceeded. Pain from one structure may also alter feedback from other structures. In the absence of cartilage pain receptors, excessive force may be applied to damaged cartilage without the ability to promote adequate protective responses.

Aging itself is associated with gradual physiologic changes in cartilage matrix, loss of repair activity of chondrocytes, and eventual development of degenerative joint disease. This is most commonly noted in people over 40, and affects mostly large joints like the hip or knee that are exposed to repeated loading (Felson, 1994, Ex. 26–544). Felson (1988, Ex. 26–114) postulated the following reasons for age-induced degenerative joint disease: metabolic changes in cartilage increase susceptibility to fatigue fracture, bone adjacent to damaged cartilage becomes increasingly stiff from microfractures, and declining muscle mass and tendon strength decrease protective shock absorbency.

At times, it can be difficult to distinguish degenerative changes caused by age from those caused by work, although many studies have demonstrated increased rates of osteoarthritis in certain working populations (see Appendix I, Ex. 27–1), and there are consistent pathogenic explanations to link work conditions to some degenerative joint diseases. Potential mechanisms include damage to subchondral tissue from excessive, impulsive, or repetitive joint loading; impaired cartilage matrix metabolism; reactive ossification and cartilage thinning; reactive bone deposition; and disturbed repair processes.

3. Vibration

Vibration is traditionally divided into whole-body vibration, particularly pertinent for seat design and transportation, and segmental vibration, affecting the hand and arm. In the latter case, health effects are usually related to energy transfer to the upper extremity from either powered tools or from stationary sources producing oscillatory vibration, such as mounted drills and pedestal grinders. Because vibration is a complex physical factor, lending itself to quantitation and modeling, and because it produces distinct and reproducible effects on blood vessels and nerves, there are parallels to noise in the formality of measurement methodology.

a. Whole-Body Vibration. Whole-body vibration can affect skeletal muscle and predispose an individual to work-related low-back pain. Etiologies for this can include bursts of cyclic muscle contraction, muscle fatigue, decreased ability of fatigued muscles to protect spinal structures from loads, continuous compression and stretch of structures, decreased blood flow, and altered neuropeptides (Brinckmann, Wilder, and Pope, 1996, Ex. 26–418; Friden and Lieber, 1994, Ex. 26–546; Hansson and Holm, 1991, Ex. 26–134; Seidel, 1988, Ex. 26–1003). Whole-body vibration, especially seated vibration, has been associated with the development of low-back disorders (Damkot *et al.,* 1984, Ex. 26–1121; Frymoyer *et al.,* 1983, Ex. 26–950; Kelsey and Hardy, 1975, Ex. 26–855; Bernard and Fine, 1997, Ex. 26–1; Troup, 1988, Ex. 26–1021). Several mechanisms have been postulated. These include microfractures at vertebral endplates, vasospasm and decreased blood flow, tissue fatigue from mechanical overload and stretching of spinal structures, and ultrastructural changes in the spinal nerve root dorsal ganglion with biochemical alterations involving pain-inducing neuropeptides (Hansson, Kefler, and Holm, 1987, Ex. 26–134; Hirano *et al.,* 1988, Ex. 26–140; Kazarian, 1975, Ex. 26–379; Keller, Spengler, and Hansson, 1987, Ex. 26–290; McLain and Weinstein, 1994, Ex. 26–1347; Pope *et al.,* 1984, Ex. 26–440; Seidel and Heide, 1986, Ex. 26–672; Seroussi, Wilder, and Pope, 1989, Ex. 26–205).

Radiographic and pathologic changes have been noted in human subjects exposed to whole-body vibration (Frymoyer *et al.,* 1980, 1983, Exs. 26–707 and 26–950; Kelsey, 1975, Ex. 26–1134; Pope *et al.,* 1991, Ex. 26–1305; Wilder *et al.,* 1982, Ex. 26–694). Christ and Dupuis (1966, Ex. 26–134) evaluated radiographic lumbar spine findings for tractor operators. As the annual number of hours of operation increased, so did the prevalence of x-ray changes. Changes were observed in 61% of operators who drove for less than 700 hours per year, 68% in those who drove for 700 to 1,200 hours per year, and 94% in those who drove for over 1,200 hours per year. The small number of subjects weakened the study. Other studies, though, have reported similar associations of driving time, symptoms of low-back disorder, and radiographic abnormalities of the lumbar spine (Fishbein and Salter, 1950, Ex. 26–267; Seidel and Heide, 1986, Ex. 26–672). Findings reported with increased frequency include reduced disc height, facet arthrosis, spondylosis, Schmorl's nodules, and spondylolisthesis. It has been pointed out that these studies have been retrospective, and some lack adequate controls (Hansson and Holm, 1991, Ex. 26–134). Unfortunately, many heavy-equipment operators and fork truck drivers are exposed to

a number of additional factors that increase disc stress, including seated postures, kyphotic postures, twisting, and whole-body vibration (Dupuis, 1994, Ex. 26–847). These probably accounts for the premature onset of degenerative disc disease in these workers.

The natural resonance frequency of the human lumbar spine in the seated position is in the range of 4 to 6.5 Hz (Magnusson *et al.*, 1990, Ex. 26–166; Wilder, Pope, and Frymoyer, 1982, Ex. 26–694). This is similar to the vibration characteristic of many motor vehicles. Whole-body vibration imposes several motions on the body and the spine, including impact, translation, and rotation. Within the natural frequency range, one animal in-vivo study demonstrated that disc pressure and axial and shear strain from vibration can increase 2 to 3 times (Hansson *et al.*, 1987, Ex. 26–134). The significant increase of spinal loading from vibration in the natural frequency has the consequence of exacerbating the amount of disc shrinkage noted after simple sitting. This has been demonstrated in human subjects using continuous measurement of the spine (Kazarian, 1975, Ex. 26–379; Magnusson *et al.*, 1990, Ex. 26–166). As frequency increases within the range of 0 to 15 Hz, stiffening of the spinal structure is noted in normal human subjects (Wilder, Pope, and Frymoyer, 1982, Ex. 26–694). Shifting to positions of mild lateral spinal flexion transiently decreases stiffness, but this posture imposes other mechanical disadvantages, such as paraspinal and abdominal muscle fatigue (Wilder, Pope, and Frymoyer, 1982, Ex. 26–694). Brinckmann *et al.* (1987, 1988, Exs. 26–84 and 26–1318) performed in-vitro experiments and noted that repeated cyclic loading of vertebral bone, as opposed to single loading events, reduced the strength of the material. They suggested that the resulting endplate fractures were a possible mechanism of later disc injury and low-back pain.

Vibration has additional effects on the erector spinae muscles, with observations of greater myoelectric activity and fatigue (Seidel and Heide, 1986, Ex. 26–672; Seroussi, Wilder, and Pope, 1989, Ex. 26–205; Wilder, Pope, and Frymoyer, 1982, Ex. 26–694). Johanning (1991, Ex. 26–1228) observed that subway operators experienced trunk muscle fatigue after being exposed to whole-body vibration for 1 hour. Pope *et al.* (1984, Ex. 26–440) also believe that the fatigue of paraspinal muscles, ligaments, and discs contributes to low-back pain associated with exposure to whole-body vibration. Progressive muscle fatigue limits the ability of skeletal muscle to protect spinal structures. Additional spinal loading can also result when the muscle response diverges out of phase with the vibration input (Seroussi, Wilder, and Pope, 1989, Ex. 26–205).

The physiologic result of vibration in the natural resonance frequency is structural failure. This occurs first in the vertebral end plate, adjacent spongy bone of the vertebral body, and the intervertebral disc (Keller, Spengler, and Hansson, 1987, Ex. 26–290). Hirano *et al.* (1988, Ex. 26–140) demonstrated that blood flow decreased in the rabbit intervertebral disc exposed in vivo to vibration. Porcine intervertebral disc experiments have shown that solute transport is also disrupted (Holm and Nachemson, 1985, Ex. 26–1374). Both of these effects are likely to precipitate disc degeneration because of disturbed metabolic activity, as discussed earlier. McLain and Weinstein (1994, Ex. 26–1347) studied ultrastructural and neuropeptide changes in the rabbit lumbar spine dorsal ganglion exposed to whole-body vibration at amplitudes and frequencies similar to those of motor vehicles. On electron microscopy, the group exposed to vibration had more significant findings of nuclear clefting, mitochondrial, rough endoplasmic reticulum, and ribosomal changes relative to controls. The authors suggested that this may provide an anatomic link between the clinical observation of increased back pain and the biochemical alterations involving pain-related neuropeptides.

b. Hand-Arm Vibration. Disorders resulting from hand-arm vibration are the sole subject of the cited epidemiologic studies on vibration. Outcomes involving measurable neurological and arterial dysfunction have taken precedence over pain and function, in marked distinction to more clinically appreciated musculoskeletal diseases. In 1986, the International Standards Organization published methods for measuring vibration and controlling its exposure—ISO 5349 (1986, Ex. 26–1301). The approach was adopted by the American National Standards Institute in ANSI S3.34 (1986, Ex. 26–1402). This accepted approach to measurement reflects the technical feasibility of characterizing the vibratory qualities of hand tools. Vibration is measured in terms of the frequency distribution of oscillations; the direction, velocity, and acceleration of those oscillations; and the impulsiveness, or force range (amplitude), expressed in each impact cycle (Starck and Pyykko, 1986, Ex. 26–678; Maeda *et al.*, 1996, Ex. 26–562). Each of these physical characteristics has a bearing on symptoms and tissue injuries that may occur, particularly in the palms and digits, but also more proximally in the shoulder and neck.

In the field of hand-arm vibration, exposure measurement and specialized disease testing have produced highly evolved, methodologically detailed, and technically sophisticated approaches. These have few equivalents in the general occupational health literature, and none in the area of soft tissue injury. The industrial control of hand-arm vibration is based on the reduction of the most prominent sign and symptom complex, cold-related finger blanching or Raynaud's phenomenon. The pioneering occupational medicine physician Alice Hamilton first described this phenomenon in the United States, among Indiana quarry workers using air-powered tools (Hamilton, 1918, Ex. 26–1401). By 1960, more than 40 studies had been published (Cherniack, 1999, Ex. 26–1354). NIOSH reviewed the available epidemiology in 1989 and 1997 (NIOSH, 1989, Ex. 26–392; Bernard and Fine, 1997, Ex. 26–1) and found overwhelming evidence of a strong dose effect between duration and intensity of vibration exposure and the onset of acquired Raynaud's, known as VWF. Arterial hyper-responsiveness and impaired vasodilation following cold challenge are also characteristics of vibration white-finger (VWF). In some studies, more than 70% of an exposed workforce evinced signs and symptoms of local vasospasm in the digits of the upper extremity, most often measured by recording finger systolic blood pressure and digital temperature stability in the setting of cold challenge (Bovenzi, 1993, Ex. 26–1280). Although a major mechanism of vibration-induced vasospasm seems attributable to local autonomic dysfunction (Gemne, 1994, Ex. 26–1320; Ekenvall and Lindblad, 1986, Ex. 26–462), a more generalized co-morbid vascular pathology may also contribute to hand symptoms and impaired function. Finger biopsies of workers heavily exposed to local vibration have shown signs of significant endothelial injury (Takeuchi *et al.*, 1986, Ex. 26–681). Increased free radical formation and elevated leukotriene B4 levels, both indicators of atheromatous injury, are observed concomitants of vibration exposure (Lau, O'Dowd, and Belch, 1992, Ex. 26–480). Overall, a satisfactory pathophysiologic model for occupational Raynaud's has been elusive.

Over the past two decades, numerous investigators have noted that neurological symptoms, including paresthesias, dysesthesias, and loss of fine motor skills among workers using air-powered tools, are even more common than vascular effects (Pyykko, 1986, Ex. 26–662; Ekenvall and Lindblad, 1986, Ex. 26–462; Futatsuka, Inaoka, Ueno, 1990, Ex. 26–547; Letz *et al.*, 1992, Ex. 26–384). It has often proven difficult to localize clinical neuropathologic symptoms to a precise anatomic locus. Accordingly, there has been considerable attention in the vibration literature to differentiating more proximal entrapment neuropathies such as CTS from distal small fiber nerve injuries in the digits (Pelmear and Taylor, 1994, Ex. 26–880; Wieslander *et al.*, 1989, Ex. 26–1027), and from more diffuse axonopathies (Farkkila *et al.*, 1988, Ex. 26–947). In the past 15 years, most investigators have recognized that small fiber injury to fingertip nocioceptors is distinctly more common than CTS in vibration-exposed workers, that electrodiagnostic studies are insensitive measures of this type of injury, and that quantitative sensory testing is essential if unnecessary carpal tunnel surgery is to be avoided (Miller *et al.*, 1994, Ex. 26–303; Pelmear and Taylor, 1994, Ex. 26–880). These tests, particularly measurement of vibrotactile thresholds, have consistently demonstrated deficits in perception in symptomatic and asymptomatic patients exposed to vibration (Flodmark and Lundborg, 1997, Ex. 26–370; Virokannas, 1992, Ex. 26–1355; Cherniack *et al.*, 1990, Ex. 26–1116). They also have shown that subjective deficits in hand functions correlate well with raised sensory thresholds (Virokannas, 1995, Ex. 26–891). The contribution of small fiber injury to deficits in touch and temperature recognition is consistent with the observation that the tissues of the digit and palm absorb well over 90% of transmitted energy from a conventional vibrating tool. The importance of small fiber nerve injury is reflected in current use of terms to characterize the health effects of vibratory hand tool exposure. The historical term ''vibration-induced white finger'' reflects the traditional focus on vasospastic symptoms. In 1987, a consensus panel meeting in Stockholm coined the term hand-arm vibration syndrome (HAVS) to give separate and equal weighting to neurological symptoms (Gemne *et al.*, 1987, Ex. 26–624).

The prominence of digital vasospasm and small fiber nerve injury in HAVS, as an outcome of vibration exposure, does not preclude other potentially important vibration-related health effects in tissues of the upper extremity. The CTS, in particular, has been recognized for its prevalence and severity in workers using pneumatic tools (Koskimies *et al.*, 1990, Ex. 26–973; Chatterjee, 1992, Ex. 26–942). Uncertainty exists, however, over the relative contributions of direct energy transfer to nerve tissue from the vibrating tool and secondary pathophysiologic or biomechanical responses to vibration that might provoke myelinated nerve injury. For example, EMG determined that muscle activity in the finger flexors, but also in the trapezii, has been affected by different qualities of vibration as well as by arm position. This is amplified in the setting of powered tools, such as nutrunners and fasteners, that create predominant biomechanical exposures other than vibration (Freivalds and Eklund, 1993, Ex. 26–116; Radwin, VanBergeijk, and Armstrong, 1989, Ex. 26–519). In these settings, more traditional ergonomic considerations, such as grip force, posture related to work surface, and duration of the torquing phase, have played a role in reported discomfort and EMG activity (Rohmert *et al.*, 1989, Ex. 26–999).

For the purpose of recognizing work-related health effects associated with vibration, it is useful to consider several pertinent features of vibratory exposure:

- Vibration is a physical factor, expressible in precise units: frequency in Hz, acceleration in m/sec^2 or G's, and cycles in milliseconds. This offers highly accessible measurement with available instrumentation, principally accelerometry and frequency spectrum analysis.

- Vibratory characteristics are highly tool-specific. Chainsaws and drills, for example, are primarily oscillatory and continuous; impact wrenches and rivet guns have large physical displacements and are highly impulsive; tools such as nutrunners have major non-vibratory biomechanical components. Thus, simple generic measurements (weighted acceleration, for example) may not capture the extent of a potential tool-specific hazard.

- Vibration can be quite well characterized as an extrinsic exposure, but health effects are the direct result of altered physiology that occurs entirely on the other side of the hand-tool interface.

Appreciation of these properties is essential for hazard identification and medical management, because significant patterns of disease have occurred in exceptional settings or tool applications that are not necessarily predictable from published standards and advisory documents. Frequency, direction of vibration, and arm and hand position all have an effect on impedance to and absorption of vibration energy (Burstrom, 1997, Ex. 26–609; Kihlberg *et al.*, 1995, Ex. 26–755). Push and pull, as well as grip force, affect transmission, and are in turn altered by the characteristics of vibration, including its impulsiveness and frequencies (Keith and Brammer, 1994, Ex. 26–1324; Griffin, 1997, Ex. 26–373).

Perhaps the most problematic area involves high-impulse acceleration. The ISO-and ANSI-weighted curves treat all vibration as harmonic, ignoring impact forces and instantaneous peak accelerations that can exceed 105 m/sec^2. Starck (1984, Ex. 26–677) noted that the dramatic reduction in vascular symptoms occurring with the introduction of anti-vibration chainsaws in the 1970s was better explained by the flattening of high transient accelerations than by a reduction in root mean square (RMS). In addition, the consistent underestimation of vascular symptoms by ISO 5349 for pedestal grinding and stone cutting was better accounted for when high-peak impulsivity was factored into the exposure model (Starck and Pyykko, 1986, Ex. 26–678). This is consistent with, but does not fully explain, the high prevalence of Raynaud's in platers and riveters, who use high-impulse tools only a few minutes per day (Dandanell and Engstrom, 1986, Ex. 26–614; Engstrom and Dandanell, 1986, Ex. 26–620; Burdorf and Monster, 1991, Ex. 26–454).

A similar problem arises in the setting of tools that oscillate at very high frequencies, such as small precision drills and saws. Most measurement protocols exclude frequencies that exceed 1500 Hz. Nevertheless, neurologic (Hjortsberg *et al.*, 1989, Ex. 26–1131) and vascular symptoms (Cherniack and Mohr, 1994, Ex. 26–1341) have been highly concentrated in select populations that use these types of tools.

Another area of importance is the occurrence of neck and shoulder pathology in workers using highly impulsive tools (Viikari-Juntura *et al.*, 1994, Ex. 26–873; Kihlberg *et al.*, 1995, Ex. 26–755). This is a complex area, particularly since the most common shoulder diagnoses—impingement and rotator cuff tendinitis—are clinically useful but without very specific pathophysiologic meaning. In the following epidemiologic review (Appendix I, Ex. 27–1), the neck, but not the shoulder, is shown to be associated with a vibration-

related pathology. The separation of biomechanical, physiologically adaptive, and vibration-specific factors is especially difficult for the neck and shoulder. Scapular stability and posture are the heart of large-muscle activation sequences involving efficient distal muscle group movement (Mackinnon and Novak, 1997, Ex. 26–1309). Moreover, static shoulder posture, important for tool stabilization, is an important contributor to early arm fatigue (Sjogaard *et al.*, 1996, Ex. 26–213). Finally, the quality of a vibratory stimulus (continuous or discrete) has significant impacts on efferent recruitment and firing (Maeda *et al.*, 1996, Ex. 26–562). The combined effects of this complexity are not easily modeled. This is all the more reason why neck/shoulder symptoms should be carefully scrutinized when a power tool is part of the exposure background. It may prove difficult in practice to distinguish neck/shoulder symptoms that have their origins in strictly biomechanical processes from vibration-induced injuries. However, there is sufficient evidence in support of an etiology to merit intervention.

The consequent injuries to blood vessels and nerve fibers from vibration are well known. When biomechanical and other ergonomic factors complicate exposures, particular attention should be paid to the tools in use, patterns of use, and specific symptom presentations.

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